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Editor
ROY WALDO MINER

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INDUSTRIAL ENZYMES—PRODUCTION AND USE†

By LEWIS B. LOCKWOOD‡

Enzymes of microbial origin, commercially available in forms separated from their parent organisms, have found wide use in extremely diverse industries during the last 50 years. The well-known enzyme preparations that are of animal or vascular plant origin, such as pancreatin, pepsin, malt, ficin, papain, and others, are outside the scope of this discussion. Further, the excellent work done by the Seagram group, the Northern Regional Research Laboratory, and the Iowa State College group is excluded from this paper since the product as used is not ordinarily separated from the parent organism. Having thus limited the scope of this discussion, I shall attempt to summarize briefly the methods of production and to mention some of the uses of the commercially available enzymes and the characteristics upon which these uses depend. In referring thus to enzymes, I speak of complex systems often containing a great variety of enzymes, but characterized or marketed on the basis of potency of one or two of their constituents.

There are several classes of enzymes falling within the limitations imposed here. Historically, and indeed also in terms of market volume, the hydrolases are the most important of these. The hydrolases include such well-known enzymes as the amylases, pectin methyl esterase, lipases, invertase, *etc.* Proteases have assumed considerable importance, and the oxidases have recently come into the food manufacturing field and show considerable industrial promise. Several other enzymes have appeared within the last few years in relatively small volume. A number of reviews of enzyme technology have appeared in the last few years.[6, 7]

The earliest commercial exploitation of microbial enzyme preparations in this country was the production of an alpha amylase of *Aspergillus flavus-oryzae* group molds by Dr. Jokichi Takamine. Alpha amylases are found in germinated cereal grains, fungal and bacterial species, and animal tissues. These all differ from the beta amylase of ungerminated cereal grains in a number of major respects, and their uses are related to these characteristics. Most important of these is the fact that alpha amylases cause a more or less random splitting of the polyhexose chains, yielding glucose and maltose and a mixture of dextrins of various chain lengths, while beta activity yields primarily maltose by terminal disintegration of chains. The random breaking of chains of glucose units results in a rapid reduction in viscosity of starch pastes or solutions.

Today, there are quite a number of amylase preparations on the market, each filling a somewhat different requirement in highly varied industries. The fungal amylases in this series are made by inoculating a solid medium such as wheat bran with a culture of *Aspergillus oryzae* or *A. flavus*. The inoculated material is then spread in trays, in a layer 1–1.5 inches thick, and incubated overnight. The temperature, humidity and ventilation of the incubation chambers are carefully regulated. The following day, the moldy bran is placed in percolators, and the enzyme is extracted with aqueous solvent or with a second extract from the previous day's batch. Automatic machinery for sterilizing, inoculating, loading, and handling trays has been developed.[5] The trays are washed by passing on a conveyor through jets of hot water, and then passed over a gas flame to sterilize them. Further on, the conveyor passes under a loading feed pipe, and the inoculated bran is placed in trays and spread automatically. In the loading feed lines are means of apportioning the inoculum and mixing it throughout the cooked bran. After filling the trays, the conveyor moves them through incubation tunnels at such a rate that, when they emerge, the fermentation is completed. The fermented bran is automatically dumped into hoppers and fed into percolators for the enzyme extraction. The aqueous extract is added to alcohol to precipitate the enzyme, and the precipitated material is collected by centrifugation. Since most enzymes are sensitive to the precipitating agents used, the precipitant is usually removed as rapidly as possible. The dry powder

* A paper by Norman F. Conant, entitled Problems Concerning Nomenclature of the Dematiaceous Fungi Pathogenic to Man, was presented at a meeting of the Division on October 24, 1952. No abstract of this paper has been received.

† This paper was presented at the meeting of the Division on April 25, 1952.

‡ Takamine Laboratory, Inc., Clifton , New Jersey.

is then diluted to standard market potency by the addition of suitable buffer salts, activators, stabilizers, and inert material.

Similar procedures have given satisfactory enzyme preparations when the organisms used were *Aspergillus niger*, *Rhizopus oryzae*, or *Bacillus subtilis*.

Considerable research has been devoted to the production of amylases in culture submerged in liquid media. Efforts to obtain a good dry powdered fungal amylase preparation having the same balance of enzymes as is obtained from bran cultures have generally proven unfruitful.

Bacterial amylase manufacture has been more amenable to deep-tank procedures, although here, also, shallow cultures are the most reliable. Three different types of culture have been used for the commercial production of bacterial amylase: the bran method, as described for fungal amylase; the shallow-liquid-culture method, in which the organism grows as a pellicle on the surface of the solution; and the deep-liquid-culture method, in which the organism grows dispersed in a deep, agitated and aerated culture solution.

In the liquid surface pellicle method,[1] shallow pans are arranged around a central shaft in a tank. Sterile air is blown through the tank over the pellicles and, after a suitable time, the cultures are harvested by rapidly rotating the shaft, forcing the liquid out against the walls of the tank to drain to the bottom and be drawn off.

There are certain advantages to be found in each of the three types of culture. In the bran process, the same equipment may be used for the production of fungal amylase. It is obvious that in both the shallow-layer methods contaminants are localized while, in the agitated-deep-tank process, contaminants are quickly spread throughout the entire culture. On the other hand, there is less danger of introducing contaminants in the two liquid processes than in the bran process.

If contamination were the only serious difficulty in operating the deep-tank process, the solution would be relatively simple, and the deep-tank process would supercede the other two culture types. Good sanitation and sound construction will solve such problems, but the major difficulty encountered in the process is not contamination with foreign organisms; it is culture variability. For manufacturing use, a variant form must be considered as a contaminant. If a variant form appears in a still culture, it does not influence the other cultures in the fermentation chamber. If, however, it appears in an agitated aerated culture, the variant may quickly outgrow the original, and even replace it completely within a couple of hours. This can be explained only on the basis of population changes on a massive scale, and not on the basis of growth from a single variant cell.

By appropriate selection of culture media and conditions, it is possible to reduce the tendency of the bacilli to vary. Salt, amino acids such as D-alanine,[2] and metallic ions[3] have been used at one time or another to control bacterial variation. Cultures can be maintained vegetatively through many generations in apparent stability on such media. If such stabilized cultures are transplanted to media lacking the stabilizing agent, massive population shifts of type may occur suddenly. This may perhaps be due to a suppression of the variation response, even though the genetic change has already occurred.

We shall now consider how this affects the commercial operation of a fermentation. The cost of the contents of a single fermentor may be so great that loss of a batch may be very serious. We cannot afford to run the risk of losing a batch through the use of faulty inocula. Consequently, most manufacturers have developed extensive inoculum-control laboratories for the stabilization and evaluation of cultural lines. If a stabilized culture is used that has developed a large masked or unexpressed accumulated variation, conventional inoculum-control methods may be quite misleading. Rapid shifts in population are not readily detected in small-scale operation, but can be disastrous in full-scale manufacturing. The fact that superficial evidence of variation is often masked may make the control tests very unreliable in estimating inoculum quality and greatly increase the difficulty of obtaining uniformity of yield and quality of product.

We shall now consider briefly the uses of these microbial amylases. Wherever rapid reduction in the viscosity of starch pastes is desirable, or the rapid solubilization of starch is necessary, microbial amylases are usually the enzyme of choice. The uses for which an enzyme is chosen may depend, however, on what other enzymes are present in the complex mixture. Thus, a commercial enzyme, Clarase Pectin, lacks pectin methyl esterase and polygalacturonase. Consequently, it is widely used in the clarification of pectin products derived from apple pomace. The starch content of pectin from untreated apple pomace

would result in cloudy jellies. Other amylases made by other strains of the *A. flavus-oryzae* group contain these pectin destroying enzymes as contaminants and, consequently, cannot be used for this purpose.

A new development in the use of the amylase of *A. oryzae* is the incorporation of this enzyme in bread doughs for the improvement of dough-handling properties, texture, and stability of the bread. The recent rejection of chemical bread softeners and approval of malt flour and *A. oryzae* amylase in the new Bread Standards by the United States Food and Drug Administration has brought the fungal amylase strongly into the baking industry. For this use, an enzyme preparation must not be proteasefree, as a certain amount of mellowing of the gluten is desired. It must also attack the starch grain, produce considerable maltose, but not accumulate too much dextrins. If dextrins accumulate in too large a quantity, or if the gluten is broken down too far, a sticky and unsuitable bread loaf results. Consequently, the enzyme preparations from only a few strains of *A. oryzae* tested are suitable.

An amylase produced by *A. flavus* finds use in the manufacturing of noncrystallizing sweet syrups from starch after mild acid hydrolysis. The preparation contains little maltase activity, and yields a relatively large percentage of low-molecular-weight dextrins and maltose, which interfere with glucose crystallization.

The amylase of *Bacillus subtilis* is of special industrial interest on account of its remarkable stability to heat. The enzyme is fully active at 95°C. and retains considerable activity even after boiling five minutes in the presence of starch. The availability of this thermostable enzyme permitted the textile industry to change the desizing of warp yarns, from low-temperature, slow, batch-type operations to high-speed, continuous, high-temperature desizing. Starch mildly attacked by this enzyme is also widely used in the sizing of fabrics and paper.

The pectin-hydrolyzing enzymes commercially available are mixtures of enzymes containing two significant constituents, pectin methyl esterase and polygalacturonase. The names pectase and pectinase have both been used for each of these enzymes and are, therefore, ambiguous and should be abandoned.

These enzymes are manufactured by either the shallow-tray or the deep-tank method. Since these enzymes are adaptive, that is, occur only when called forth by their substrate, the *Penicillia* that produce them are grown on pectin-containing media. Any source of pectin appears suitable if toxic substances are not present; sugar beet cossets, a waste product of sugar refining, are a suitable substrate for the production of pectic enzymes.

Pectin is a polymer of galacturonic acid, in which most of the carboxyl groups are esterified with methyl alcohol. Pectin methyl esterase hydrolyzes the ester linkages, producing pectic acid and methyl alcohol. The insoluble pectic acid so produced in fruit juices is removed by filtration. Polygalacturonase hydrolyzes the glycosidic bonds between the galacturonic acid units of pectic acid and, thus, reduces the number of hexuronic units in the polymer, and produces some free galacturonic acid. Polygalacturonase is of value whenever it is desirable to reduce the viscosity of pectic materials.

Although widely used in the clarification of apple juice, none of the commercially available preparations of pectic enzymes completely solve the problem of delayed haze formation. In untreated juices, the pectin methyl esterase naturally present in the apple juice will cause a slow precipitation of pectic acid, yielding a cloudy juice. If the enzyme is added to the juice and the pectic acid filtered off, an additional haze formation occurs after the juice has stood for about six weeks. This is not affected by the pectic enzymes.

The commercially available microbial proteases are of bacterial origin. The protease of *Bacillus subtilis* is manufactured in essentially the same way as is the amylase. The two enzymes commonly occur together, but protease destruction occurs in the culture earlier than amylase destruction. This is true in either shallow-layer or deep agitated cultures. The strains of *Bacillus* that give the best yields of amylase are not necessarily the best producers of protease.

Bacterial protease finds its principal use in the removal of spots from soiled clothing or upholstery by dry cleaners. In fact, this business exceeds one million dollars annually. The enzyme is also used in the bating of hides and removal of gelatine sizes.

The advent of penicillin gave rise to the need for an enzyme to inactivate the antibiotic in order to test the sterility of ampoules. Penicillinase inactivates penicillin by conversion to penicilloic acid. The enzyme is manufactured by the aerated agitated cultures of *Bacillus cereus* or *B. subtilis*. It is precipitated from the clarified culture liquor

with an organic precipitant and is marketed in bulk as the dry enzyme concentrate or in ampoules of definite standard potency. It now finds its greatest use in penicillin production control where the Ford rapid-assay method is used.[4]

Last year, a new enzyme preparation, which contains a mixture of two enzymes, streptokinase and streptodornase, was placed on the market. These enzymes are made by hemolytic streptococci grown in aerated culture. The mixture of enzymes serves in the debridement of surfaces covered with necrotic tissues or pyogenic membranes but does not attack healthy tissue. It is used in the dressing of wounds and burns.

Some enzyme systems have to be extracted from the living cells, as they are not found in the culture substrate in sufficient quantity for manufacturing requirements. Three important enzymes in this group are invertase, catalase, and glucose oxidase. The general procedure for the recovery of intercellular enzymes involves the destruction of the cell structure, extraction of the enzyme with aqueous solvent and precipitation. Invertase is obtained from the yeast by autolysis under toluene, followed by extraction with water or buffers, filtration to remove cellular residues, and precipitation with alcohol.

Catalase, an enzyme that decomposes hydrogen peroxide, yielding water and nascent oxygen, appeared on the market several years ago. At least one catalase preparation now available is obtained by growing a mold in aerated agitated culture. The microbial catalase preparations, as marketed, are high-potency liquid preparations or diluted products directed toward special markets.

Catalase is used in the bleaching and dying of furs and in the bleaching of human hair to remove residual hydrogen peroxide, which causes undesirable color changes if not completely removed. It is also used in the manufacture of surgical gut.

One of the most recent additions to the group of commercially available enzymes is glucose oxidase. It is obtained, associated with catalase, from aerated agitated cultures of *Aspergillus niger*. If such an enzyme is applied to a glucose-containing substrate, the glucose is oxidized, and gluconic acid or one of its salts accumulates in the substrate. This reaction finds use in the desugarizing of egg whites and whole eggs prior to drying, since the undesirable flavors and color of dried egg whites are usually due to reactions involving the glucose. If the enzyme system is permitted to react with an excess of glucose in the presence of a limited amount of air, as in a bottle of beer or a can of fruit, oxygen is removed from the air and the solution and combined with glucose. The oxygen tension is thus reduced and the occurrence of those undesirable flavor changes due to oxidation is prevented.

I have tried to summarize briefly the principal commercial classes of enzymes now available, giving some indication of their uses and their properties upon which those uses are based. I have also tried to indicate a few of the many problems yet awaiting solution.

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SECTION OF GEOLOGY AND MINERALOGY

CALIFORNIA EARTHQUAKES*

By PERRY BYERLY†

Our first and still most important reference in the study of California earthquakes is their history as set forth in the "Catalog of Earthquakes of the Pacific Coast of the United States" and its extension, the annual "United States Earthquakes." The catalog carries us through 1928. In its latest edition, it appeared as a number (Volume 29, No. 1) of the Bulletin of the Seismological Society of America. "United States Earthquakes" is an annual publication of the U. S. Coast and Geodetic Survey. It has appeared to me that, in current times, we worship instruments a little too much. There is a tendency to neglect the keeping of an excellent historical record. When one reads discussions of the results of a study of seismograph records, one finds that many of these, based on data of, say, thirty years ago, seem much outmoded. Instruments develop rapidly. But the history, the honest detailed discussion of field observations, remains engrossing and up to date.

As in other fields of science, the measure of the value of any "laws" we may discover is their value as a means of prediction. As far as finding any law enabling us to predict when earthquakes will occur, we have not yet had much success. Perhaps we do not yet have a long enough record. The first earthquake on record in California occurred, according to the Catalog, in 1769. It is based on a story told to Father Serra by the Indians. But the higher criticism has questioned it. The Indians included in their story an accompanying eclipse of the sun that, the astronomers say, did not occur. Entries in the Catalog are naturally sparse for the early days, being based on reports of Spanish or Mexican explorers who happened to be travelling through California at the time of occurrence.

There certainly have been three very large earthquakes in California since the record began. In 1857, the San Andreas Fault, which traverses the Coast Ranges, broke in the vicinity of Fort Tejon (northern Los Angeles County now). The length of the break was perhaps two hundred miles. We cannot say from the reports what the displacement was. The damage was great at the Fort but minor at the Mission of San Fernando (now in greater Los Angeles). It was felt at least as far north as San Francisco. The physiography along the San Andreas Fault indicates that the Pacific side of the fault had been moving northwesterly relative to the continental side long before we have any history written by man. Not uncommonly, geologists have claimed evidence of displacement in tens of miles, and someone told me the other day of a recent work that claims hundreds of miles. No one can question that displacements of thousands of feet have occurred.

In 1872, the Owens Valley Fault at the eastern base of the Sierra Nevada was the seat of another great earthquake. The faulting was more or less en echelon, with the displacements on various sections being somewhat discordant. Nevertheless, displacements were large, 10 to 15 feet. The earthquake was felt throughout California and Nevada and was most destructive in the hamlet of Lone Pine. Once again, the area of the center of the shock was sparsely settled.

The San Francisco earthquake of 1906 was the third great shock. The shock is world famous because of the resulting great damage over a wide area, for the central area was well populated. It is geologically most famous because of the great fault displacement, which is unmatched before or since in historic time. The San Andreas Fault broke over 270 miles of its length, from Upper Mattole in Humboldt County to San Juan Bautista in San Benito County. The displacement was almost wholly horizontal and reached a maximum of twenty-one feet at the head of Tomales Bay. The west side moved northerly which, as the physiography tells us, has been the custom of Coast Range faults. It was the earthquake that fixed firmly in American geologic thought the idea that faulting is the cause of earthquakes. Some Americans are startled to hear that anyone doubts this. Many Japanese question it. The other idea is that faulting and earthquake waves are two rather independent results of some cataclysm at depth. The argument for grating along a fault as the source of earthquake waves lies first in its reasonableness and, second, in the observation, for perhaps a dozen cases, of faulting at the time of the shock of sufficient magnitude to suggest a reasonable source of energy.

We sometimes cite as additional evidence the

* This paper was presented at a meeting of the Section on October 6, 1952.

† Department of Geology, University of California, Berkeley, California.

general occurrence of earthquake centers along fault zones. On thinking about it, one sees a great weakness in the theory. The correlation between earthquake centers and surface fault breaks is almost nil. Although most California earthquakes center in the Coast Ranges, which are heavily faulted, there are many other faulted mountain ranges in the United States that are not the seat of frequent shocks. The observation that for almost all large earthquakes the first motion of the primary wave is distributed between compressions and rarefaction in alternating quadrants about the source indicates that the source is reacting to a couple. This strongly suggests faulting.

If one were to list the three most destructive earthquakes in California, one would include: San Francisco 1906, Santa Barbara 1925, and Long Beach 1933. The latter two were not large earthquakes in the energy contained, but they centered in populous regions.

We shall turn to a smaller region to investigate the temporal occurrence of destructive shocks. The region about San Francisco Bay has the unenviable history of having had the most shocks. Some of this is no doubt due to its longer settlement. A large part may be blamed on the presence of the San Andreas and Haywards Faults in the region. The assessment of the intensity of early shocks is difficult, because of the sparseness of written record. Up to the late 1840's, California had no newspaper. In the 1830's, when large shocks occurred, the church was being secularized and there are no church records. Old diaries are one source. After the large shocks of the 1860's, newspaper reporters looked up old men who had experienced the earthquakes of the 30's and wrote the stories. Anyone who has discussed the past with older relatives knows well that their memories and one's own do not agree in many details and that, therefore, the old people are obviously wrong. Particularly, is it troublesome to fix the date, the year of the earthquake. In the catalog appear too many large shocks in the Bay area to fit our ideas of the mechanics of earthquakes. But if we grant that one oldster was a year off in his memory, we can reduce two successive great shocks in 1838 and 1839 to one in 1838.

After much consideration, we are left with five great shocks in the area: in 1836, 1838, 1865, 1868, and 1906. It is dangerous to try to get periodicities out of so short a record. But what are geologists to do with such a cranky and erratic earth as ours? We must generalize one way or another. Consequently, there have been those who have said that 40 years was the approximate period and others have argued for a period of 70 years, discounting the earthquakes in the 60's one way or another. One suspects from the record that it was the 1838 shock that was nearest the size of the 1906 shock.

We have another approach, however. In the Report of State Earthquake Investigation Commission, Harry Fielding Reid analyzed the triangulation observations of the Coast and Geodetic Survey over many years in connection with the fault displacements. He summarized his work in Hitchcock lectures delivered at the University of California. His conclusion was that the coastal region of California west of the San Andreas Fault had been drifting north, relative to the area east of the fault, at a rate of about 20 feet in an hundred years, and that the 1906 fault break was the release of slow strain accumulated over that period. This idea of slow drift was anathema to the Geodetic people and also to the local astronomers. You can readily see why. The geodetic data on which the inferences were based were somewhat scattered and it was genius in Reid that interpreted them. For today, after many repeated and careful triangulations across the fault, the Coast and Geodetic Survey (Whitten) informs us that the drift exists and is about two inches a year near San Francisco Bay. So we have another figure. Perhaps a shock as big as the 1906 one takes a hundred years to build up and if shocks occur more often they will be smaller.

The failure of the San Andreas Fault to act reasonably has bothered some of us. If it broke for a hundred miles on either side of Fort Tejon in 1857, why was the next break not adjacent to this, instead of being well to the north? What must be the condition of strain in the region between the two breaks? As has been reported recently, a great earthquake has occurred near Tehachapi in southern California. On first reports, we all thought that this was the repetition of the 1857 earthquake at roughly the 100-year period. But field investigation as well as instrumental epicenter location suggest as a source the Bear Mountain Fault near the town of Caliente. There was cracking along the fault and, perhaps, displacements of a few feet, although that is not certain. The plotted fault is curved with a past history of probably vertical motion. Can such a shock relieve strain along the San Andreas Fault near

Fort Tejon, or does it just indicate a grave situation in the region? According to the magnitude scale, this recent shock had in it only about one fifth of the energy in the 1906 earthquake.

For many years, large networks of seismographic stations have been operated by the University of California in northern California and by the California Institute of Technology in southern California. Routinely, each locates the epicenters of all earthquakes recorded at several stations.

The fact has gradually emerged that the epicenters of the smaller earthquakes so located do not, in general, lie along the recognized fault traces of the region. The epicenters of the larger shocks do, however. Do we make errors in our locations, or is it the case that minor ruptures tend to occur in the rock body, making or utilizing minor fractures?

In conclusion, I wish to make a few remarks about the earth's surface layering. Geologists have known from a time long before seismology was recognized that the earth's surface structure is highly complex and that, no doubt, to very considerable depths. Back in 1909, A. Mohorovicic proved seismometrically that in Europe there is a major discontinuity, roughly horizontal at a depth of several tens of kilometers. The same seismic waves that establish this turn up in Japan, in South Africa, and in America. About 30 km. seems to be the average depth although, under the Sierra Nevada, I have been able to show it is much deeper. The root of these mountains casts a seismic shadow.

Since 1909, various seismologists working with more or less sparse local earthquake data have interpreted them as indicating discontinuities at higher levels. The earth has had a two-layered crust, and sometimes a three- or four-layer crust, at different times in different places. Whereas the existence of the Mohorovicic discontinuity has never been seriously questioned, although its depth has varied from 30 to 60 km.; the other discontinuities have been found by some and not by other observers. For myself, I find different ones every time I tackle new data. Currently, new men entering the field declaim that there are no intermediate discontinuities. They must remember that very many such varying from place to place are practically equivalent to none, but that, in particular cases, the data frequently demand one. I think we now can agree that there is no intermediate discontinuity that exists all over the United States at the same depth. But there is in California something funny at a depth of about 10 kilometers over much of the state. It is no doubt rough and irregular, but refraction earthquake data demand it, whether or not it is a good reflector.

The introduction into seismology of vast quantities of new data is making its effect felt, not in making us more definite, but in calling a halt to general conclusions. More than ever, seismologists are realizing that the hodge-podge of rocks we see at the surface remains a hodge-podge at least to a depth of 30 kilometers.

*SECTION OF BIOLOGY**

STREPTOMYCIN†

By W. W. UMBREIT‡

The subject of chemical structure in relation to the mode of action ought to be a rather nice one for streptomycin since, in the first place, we know the structure of the molecule and, in the second place, we know something about the mode, if not the mechanism, of action. So that all we need do is to compare the two. The structure of streptomycin (shown in FIGURE 1) also gives one a good field for action. There are two guanido groups, an inositol area, a streptose portion, which looks like a substituted desoxypentose, and then N-methyl glucosamine. Now this molecule ought to interfere with something. Its guanido groups and its streptose might disturb some nucleic acids, and the actions of inositol or glucosamine are sufficiently confused so that quite a few speculations are possible.

Nature, and chemists, tinker with this molecule some, and we find mannose added to the glucosamine to give mannosido streptomycin and a few other changes indicated on the slide. But this is

* The Section of Physics and Chemistry held a meeting on October 7, 1952, at which a paper, entitled High-Energy Physics, was read by Warren Goodell. No abstract of this paper has been received.

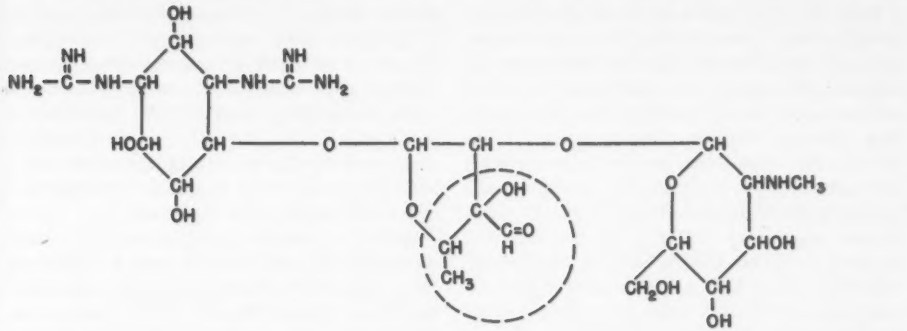
† This paper was presented as part of the Symposium on Chemical Structure of Antibiotics in Relation to Mode of Action at the meeting of the Section, October 13, 1952.

‡ Merck Institute for Therapeutic Research, Rahway, New ersey.

all. Anything else one does to this molecule destroys, for all practical purposes, its antibiotic activity. Indeed, streptidine and streptobiosaminė are not antibiotically active, separately or mixed together.

Now it would seem reasonable that a molecule as complex as this one, with as many nubs and corners, will probably interfere with something, if only the case of the "butter not suiting the works."

Streptomycin does indeed precipitate and form complexes with nucleic acids and nucleoproteins.[1] This effect was at one time mistaken for an enzyme, the so-called streptomycinase, but further study showed that the basic properties of streptomycin were responsible for complex formation with a variety of acid materials, ribose nucleoproteins, desoxyribose nucleoproteins, cephalin, oleic, palmitic, and tannic acids, *etc.*[2,3] This combination



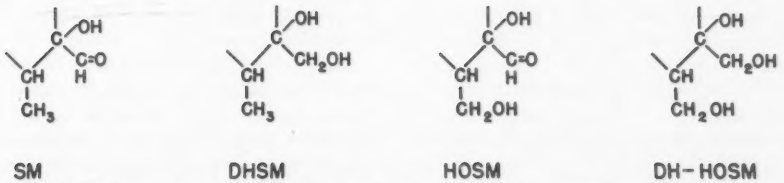


FIGURE 1. Chemical structure of streptomycin and derivatives. Abbreviations: SM = streptomycin; DHSM = dihydro-streptomycin; HOSM = hydroxystreptomycin; DH-HOSM = dihydro-hydroxystreptomycin.

Some of these interferences may be quite specific and directly related to how it kills the organism; but some will probably be due to the fact that this big awkward molecule gets in the way of some reactions we want to conduct using preparations from living material, but the living cell is not bothered by these things. We shall therefore find it necessary to look a bit critically at the observations to see whether they could be the mode of action of the drug, or whether the effects exist more in our apparatus or measurements than in the living cell.

apparently alters the surface charge of the bacteria and is probably responsible for the agglutination of some cells by streptomycin.[4] We have the strong suspicion that this complex formation is what is encountered in assays in different media, in observations on inhibition of nucleic acid metabolism, and indeed in some of the cases of amino acid metabolism anaerobically. Taken as a group, we would feel justified in eliminating those reactions that are similarly affected by streptomycin and by either streptidine or streptobiosamine. Since the latter two are not antibiotics, unless there

is a marked disparity in the concentrations required, reactions inhibited by streptidine or streptobiosamine cannot be those concerned with the antibiotic action, else these compounds would be antibiotics. Furthermore, we may eliminate several other reactions and assign to them the role of merely reflections of a related but awkward molecule getting in the way, because the concentrations of streptomycin required are so far beyond those required to inhibit growth.

These objections, however, do not eliminate the precipitation of desoxynucleic acid as a site of action of streptomycin. The concentrations of streptomycin required to form such precipitates are not too far above those inhibiting growth, but here concentration may be misleading. It is known that, within the limits of measurement, virtually all of the streptomycin remains outside of susceptible cells, and that only a very small amount penetrates.[5] Therefore, the concentration required to inhibit the cell may be considerably more than is actually required to stop the sensitive reactions, since only a small part penetrates the cell to the site of such inhibitable reactions.

Furthermore, the streptomycin–nucleic acid complexes are redissolved by various salts. Salts also tend to inhibit the action of streptomycin. The activity of various salts is not the same in the two measurements.[6] There are also some experiments on the fact that the streptomycin–DNA complex may be broken and the DNA depolymerized by the desoxyribonuclease, whereas this enzyme has no effect on the minimum level of streptomycin required to inhibit a particular organism. The more one thinks about these experiments, the less pertinent they seem to be. I really think that we cannot critically eliminate this combining power of streptomycin as a factor in its action. We have no real evidence that will strictly eliminate it. I think the chief difficulty with it is that it is so vague and so nonspecific, so that we are not satisfied with this explanation, particularly, since it leads us no further into understanding. The oxidation of ribosenucleic acid has been reported to be inhibited by penicillin and streptomycin,[7] but this seems to be a peculiarity of a particular strain of *Staphylococcus aureus* used, since others apparently do not show this phenomenon.[8]

Another set of reactions in which the chemical structure of streptomycin seems to be involved is the inhibition of diamine oxidase[9, 10] in a variety of acid-fast organisms and in some others possessing measurable amounts of this enzyme. This is apparently due to the presence of the two basic guanidine groups in the molecule. In streptomycin-resistant organisms, the diamine oxidase is not so inhibited, and the diamine oxidase of the animal is not inhibited by streptomycin. The enzyme is also inhibited by a variety of other substituted synthetic amines, as well as by pyocyanin, streptothricin, and chloramphenicol. The concentration of streptomycin required for inhibition of diamine oxidase is somewhere between 6 and 60 μg. per ml. in organisms sensitive to 1 μg. per ml. While it is debatable whether this bears a direct relation to the mode of action of streptomycin (what is one going to do with the wide array of sensitive organisms possessing no detectable diamine oxidase?), it is certainly a reaction whose inhibition is due to the particular chemical structure of the streptomycin molecule.

Looked at from one aspect, streptomycin might be considered a derivative of inositol. There are two actions of streptomycin that seem to be related to inositol: First, an inositol-utilizing mutant of a *Torula* was obtained, which was sensitive to streptomycin when grown on glucose or on inositol, whereas the parent was not.[11] The inhibition by streptomycin was relieved by the addition of glucose-1-phosphate or hexose-diphosphate. Second, it was reported that an inositol phospholipid (lipositol) antagonized the action of streptomycin[12] but, eventually, it was found that the effect was an artifact,[13] so this relationship has been discarded, at least for the moment.

Streptomycin may be written in such a manner that it would appear to exist in three parallel planes with an oxygen or carbon bridge between each two. Each of the planes would be occupied by a ring structure. I do not say that the streptomycin molecule is this way, but only that one can look at it this way. As such, it might be expected to interfere with the formation or the metabolism of ring structures. Indeed, it first seemed as if this might be the case, since it was reported to interfere with the oxidation of benzoic acid by certain mycobacteria.[14] It was later found, however, that this effect was due to streptomycin interfering with adaptive enzyme formation rather than with the oxidation of benzoate *per se*.[15] Incidentally, using other adaptive enzymes as a criterion, we find that most of the clinically important antibiotics (streptomycin, chloramphenicol, aureomycin, *etc.*) interfere with adaptive enzyme formation, but that

penicillin does not. The reaction interfered with is probably different in each case and certainly not revealed by the net result of lack of adaptive enzyme formation, which may be many steps away; I would not like to venture a guess as to how many steps it might be.

Streptomycin is thought by some to bear a relation to pantothenate or coenzyme A. I do not quite see this but am willing to grant the supposition if it leads us anywhere. *Saccharomyces fragilis* is sensitive to streptomycin when grown on β-alanine as source of Co. A, but not sensitive when pantothenate is supplied.[16] This seems to be somewhat limited to this organism, however. Pantothenate does not antagonize streptomycin in other cases.

The foregoing amply demonstrates, it seems to me, that there are several actions of streptomycin that do bear some relation to the chemical structure of the molecule. But do these actions bear any relation to how the antibiotic kills or inhibits the growth of sensitive strains? Here, one must begin to set up some standards, some criteria for determining whether or not a reaction acted upon by an antibiotic can be, or is, the important reaction in killing the organism. Three such criteria are at present in use.[17] These are:

(1) The effects observed must be evident for the antibiotically active forms of the antibiotic, but show no reaction with those derivatives of the drug that have no antibiotic activity.

(2) The concentrations of the drug required to act on the reaction must be comparable to or identical with those required to inhibit growth.

(3) The reaction inhibited must be vital to the cell.

It may be necessary to add some additional criteria, but these are the ones we are operating with at present.

There is one reaction that comes pretty close to meeting all of these criteria. We have called it the oxalacetate–pyruvate reaction.[18, 19, 20] Many other workers have found it too, and it has been measured in a variety of ways, all of them indirect. What we have said about this reaction is that it is not one of the known reactions of pyruvate or oxalacetate. It is rather curious that, in the case of both penicillin and streptomycin, one should run into reactions of apparently considerable importance that are quite unsuspected by other methods of studying metabolism. In the case of streptomycin it is a reaction mixed right in with an array of others that have been studied for years. In fact, many people feel that, because we know so much about about the reactions of pyruvate and the citric acid cycle, it is unlikely that a new reaction will be found here, and that the obvious effects of streptomycin on the system are due to its inhibition of known reactions. But no one has demonstrated any inhibition of streptomycin on any of the known reactions.

So we rest the case at the moment on an unknown reaction. Since we do not know its mechanism we cannot tell whether the chemical structure of streptomycin is involved *per se*, but we certainly do know that one did not find this reaction by looking at the streptomycin molecule and thereby deriving a mode of action. Nature still has a few tricks up her sleeve, and the mode of action of antibiotics is evidence of one of them.

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CHEMICAL STRUCTURE OF TERRAMYCIN IN RELATION TO MODE OF ACTION*

By PETER P. REGNA†

It is expedient to distinguish broadly between two methods of approach to a study of the mechanism of antibiotic action. In some cases, important basic biological information can be obtained from investigations on the inhibition of dynamic metabolic processes, without knowledge of the chemical nature of the antibiotics involved. It is the objective of a second approach to the study of the mode of action of an antibiotic, such as terramycin, to consider the effects of modifying the structural features of the substance, thus revealing the significance of individual chemical groupings on biological action.

Aside from observations that terramycin inhibits the growth of a wide variety of microorganisms *in vitro*[1] and *in vivo*,[2] the mode of action of terramycin is essentially obscure. Several investigators, however, have reported on cell inhibitory processes affected by terramycin[3] or aureomycin,[4] a closely related broad-spectrum antibiotic.[5] Of particular interest in this connection are the observations of Loomis[6] that aureomycin, in concentrations of the order of 100 γ/ml., inhibits phosphorylation without inhibiting cell respiration. Moreover, certain experiments of Gale[7] have shown that, at similar concentrations, aureomycin is far more effective in inhibiting the condensation of glutamic acid into cell protein of *Staphylococcus aureus* than it is in preventing the accumulation of glutamic acid within the cell.‡ While aureomycin differentiates between glutamate accumulation within the *Staphylococcus aureus* cell and the energy-supplying glucose fermentation, terramycin, at these concentrations, does not interfere with accumulation of glutamic acid in the cell.

More important than this, Gale[9] has pointed out further that, at much lower concentrations, terramycin and aureomycin have an even greater inhibitory effect on the synthesis of cell protein. The concentrations of these antibiotics causing 90% inhibition of protein synthesis are approximately the same as those inhibiting growth of *Staphylococcus aureus* in a medium composed of glucose, amino acids, purines, and pyrimidines. It is of interest that concentrations of these antibiotics that bring about a breakdown of protein synthesis within cells significantly activate nucleic acid synthesis.

The report of Loomis[6] that concentrations of aureomycin in the neighborhood of 100 γ/ml. will uncouple (*i.e.*, dissociate) oxidative phosphorylation in mitochondrial preparations has been confirmed by Brody and Bain.[10] At similar concentrations, aureomycin uncouples phosphorylation from oxidation in rat-liver- and brain-tissue preparations. At concentrations of about 300 γ/ml., however, both oxidation and phosphorylation are inhibited. In contrast, terramycin at these concentrations produces little inhibition of either oxidation or phosphorylation in these tissues.

The second approach is not concerned, at present, with such details of the mode of action of terramycin, but rather with the effects of structural changes in the antibiotic as measured by simple antimicrobial activity. During the course of the determination of the structure of terramycin, a host of degradation products became available that contained one or more of the structural elements of terramycin itself. A thorough examination of these may quite well permit us to draw certain limited conclusions regarding the structural features of terramycin essential to antibiotic action. Derivatives are considered inactive when they show no inhibition against *Klebsiella pneumoniae*, when added to nutrient media in concentrations of one part in 10,000. This level corresponds approximately to an activity of 0.1 per cent of that of terramycin under our test conditions.

Among the first new compounds (FIGURE 1) isolated during the course of alkaline degradation of terramycin (I)[11] were terracinoic acid (II), and much smaller quantities of isodecarboxyterracinoic acid (III),[12] terranaphthol (IV),[13] and 7-hydroxy-3-methylphthalide (V).[14] Since the last four substances all contain a hydroxybenzene ring and the sole methyl group in terramycin, they were presumed to embody the same aromatic ring. The 7-hydroxy-3-methylphthalide contains, in addition, the carbonyl group of ring C in terramycin. This substance was inactive by our test, and it was presumed to have only the activity of a simple phenol. It is apparent that this simple system is not sufficient to provide the antibiotic properties of terramycin. Terranaphthol, which is composed of both the D and C rings of terramycin, is also inactive. It should be noted, however, that the

* This paper, illustrated with lantern slides, was presented as part of the Symposium on Chemical Structure of Antibiotics in Relation to Mode of Action at the meeting of the Section of Biology on October 13, 1952.

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‡ Bacterial concentrations of penicillin inhibit the passage of glutamic acid into *Staphylococcus aureus*, so that the internal concentration decreases until exhausted by internal metabolic processes.[8]

ring C of terranaphthol, unlike that of terramycin, is aromatic and planar. Strictly speaking, a conclusion regarding its contribution to the biological activity could be valid only if the two ring systems were hydroaromatic, as in terramycin.

Terracinoic acid and isodecarboxyterracinoic acid contain, in addition to the aromatic ring of terramycin, a new 5-membered carbocyclic ring system that, we now know, results from a condensation of an unstable primary nonasymmetric precursor. Since these acids are inactive and possess none of the structural features of terramycin not disclosed in the 7-hydroxy-3-methylphthalide derivative or in terranaphthol, we need not consider them further.

Under controlled aqueous acid conditions, more deep-seated changes leading to dehydrations, to aromatic structures, and to rearrangements are brought about. In dilute acids, terramycin loses a molecule of water and rearranges to form two closely related optically active substances, α- and β-apoterramycins (VII).[15] In this rearrangement, aromatization takes place with subsequent cleavage of ring B in terramycin to yield a dihydroxybenzophthalide system. Ring A, however, remains intact, precisely as it exists in the terramycin molecule. Since the apoterramycins have

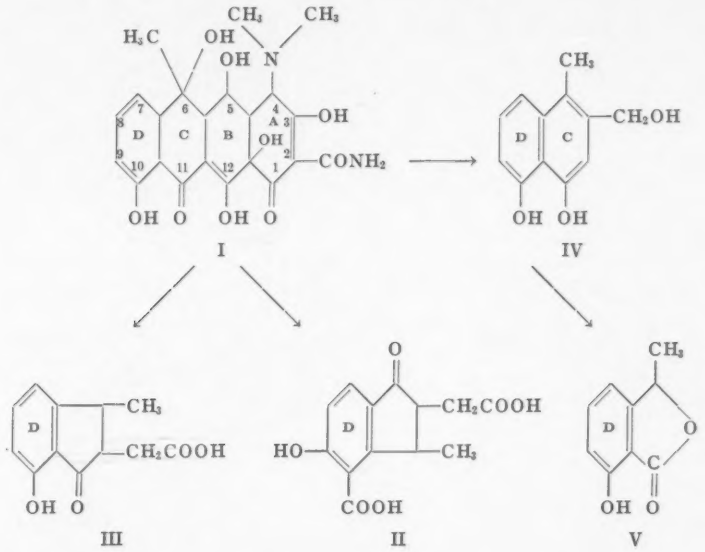


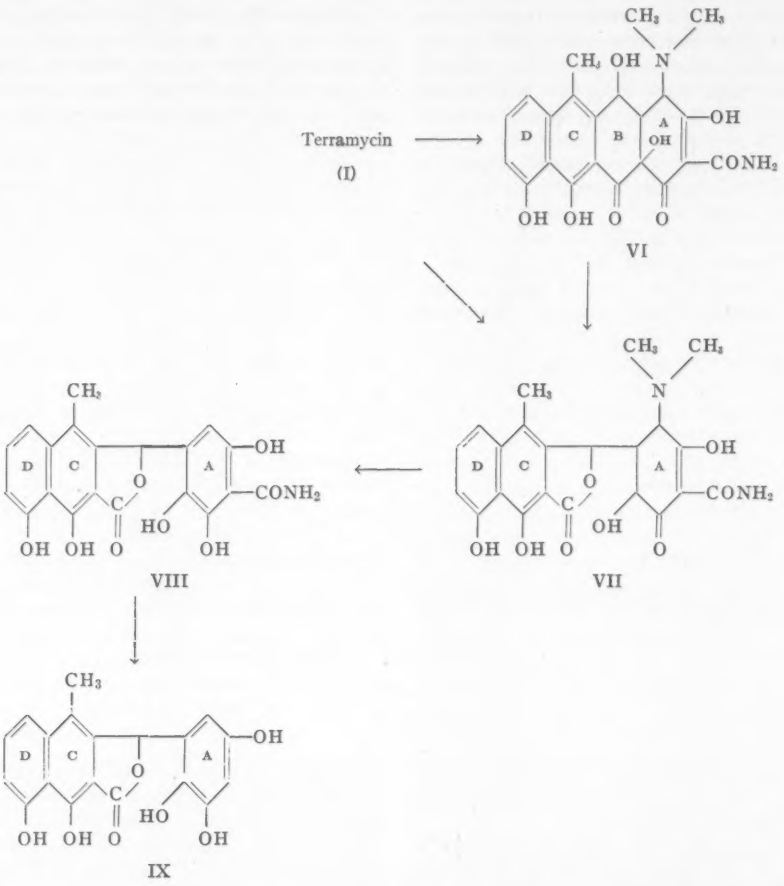
Fig. 1

An integral part of the structure resolution of terramycin was derived from a study of the acid degradation products. Anhydroterramycin (VI)[13] can be obtained from the treatment of terramycin (I) with anhydrous hydrogen chloride in acetone. The unstable anhydroterramycin (VI) differs from terramycin only in the loss of a mole of water and consequent aromatization of ring C. It does possess a biological activity of the order of one per cent of that of terramycin and, perhaps, it is significant that aromatization of ring B does not in itself destroy all antibiotic activity.

no detectable biological activity, it can be assumed with considerable justification that none of the antibacterial activity of terramycin resides in ring A by itself but, rather, that it is essential to the framework of the whole molecule. The apoterramycins differ from anhydroterramycin only with regard to ring B; the C_{12-12a} bond breaks with consequent formation of a phthalide ring. It may be inferred from this preliminary evidence that formation of a new ring B completely destroys antibiotic activity.

Over long periods of time, under conditions of their formation from terramycin, the apoterramycins lose dimethylamine to form an optically active substance, terrinolide (VIII).[16] On further

hydrolysis in hot 12*N* sulfuric acid, terrinolide loses ammonia and carbon dioxide to yield a nitrogen-free optically inactive substance, decarboxamidoterrinolide (IX).[16] IX is similar to the apoterramycins but is distinct in that ring A lacks the dimethylamine and the carboxamide groups and amine and a hydroxyl group to yield desdimethylamino-12a-desoxyterramycin (X).[17] This reduction product shows but very slight biological activity against gram-positive and gram-negative microorganisms. It would appear, therefore, that the replacement of the dimethylamino and the



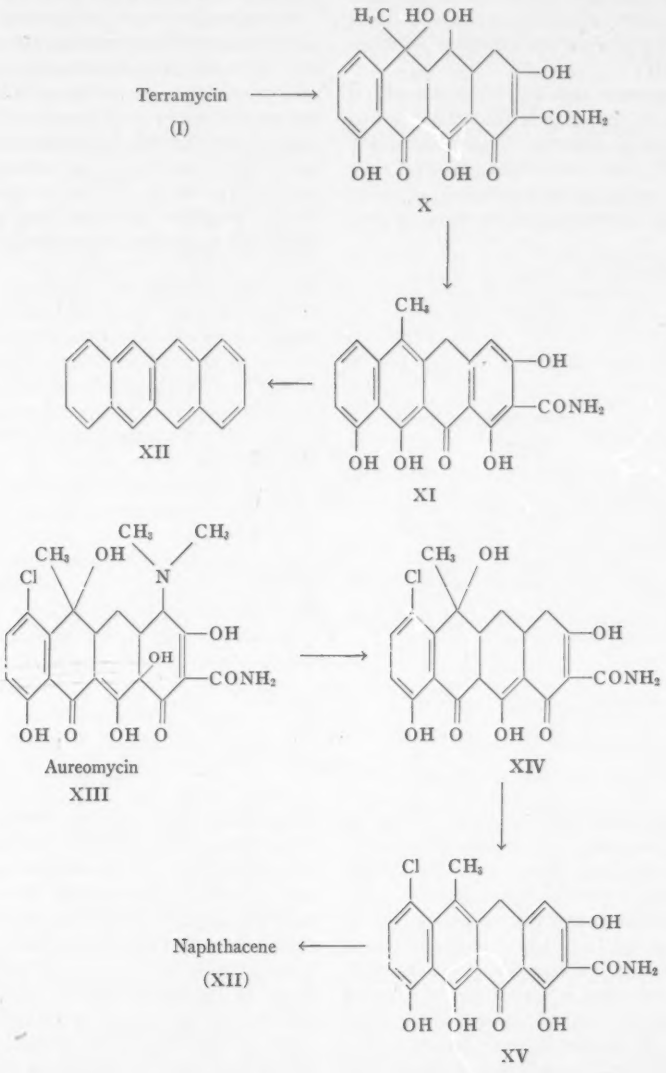
is completely aromatic. Both VIII and IX are inactive by our test, despite their polyphenolic characters.

A most important series of reactions connected with the elucidation of the structure of terramycin was the demonstration of the existence of a hydronaphthacene skeleton. These reactions were initiated by reduction of terramycin with zinc and acetic acid, with subsequent cleavage of dimethyl-

C_{12a} hydroxyl groups, by hydrogens, results in a marked decrease in antibiotic activity.

Under mild acid conditions, X loses two molecules of water, resulting in a highly insoluble orange-red colored compound, desdimethylaminoterrarubein (XI),[17] containing a totally aromatic and conjugated system. The substance appears to be inactive, despite retention of the naphthacene skeleton and its high degree of phenolic properties.

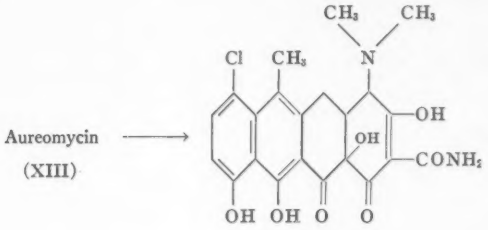
Zinc-dust distillation of XI yields the aromatic hydrocarbon naphthacene, establishing a linear tetracyclic structure for this series of substances.



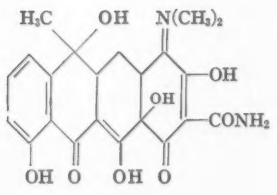
Throughout the course of our structure work on terramycin, several papers[18, 19, 20] appeared emphasizing the similarity of terramycin and aureomycin, and suggesting that the nonionic chlorine atom in aureomycin had been replaced by a hydroxyl group in terramycin. It was realized early that this deduction was in error, since low-temperature alkali fusions yielded 5-chlorosalicylic acid from aureomycin[21] and salicylic acid from terramycin.[11] If a hydroxyl group had actually been substituted for the chlorine in aureomycin (XIII),[22, 23] gentisic acid rather than salicylic acid would have been isolated from terramycin. The

ultraviolet spectrum and the acidity constants of aureomycin (pK's 3.4, 7.4, 9.2) are very similar to those of terramycin (pK's 3.5, 7.6, 9.2), indicating that the carbonyl system of terramycin is common to both.[22] Thus, terramycin and aureomycin have the relationships expressed by structures I and XIII.

The naphthacene skeleton in aureomycin is demonstrated by the series of reactions indicated for terramycin: by reduction to desdimethyldesoxyaureomycin, from which is obtained a red compound (XV) containing one molecule of water less and from which naphthacene is obtained by zinc-dust fusion.



XVI



XVII

Anhydroaureomycin (XVI),[23] obtained from the treatment of aureomycin by concentrated hydrochloric acid, has been reported by the Lederle workers[24] to have some biological activity. This substance parallels the terramycin analog (VI) by the absence of the C_6 hydroxyl, aromatization of the D and C rings, and retention of intact ring A.

To summarize, then, in correlating the structure of terramycin with antibacterial activity, it is apparent that the results of these limited experiments do not reveal the existence of the minimal portion of the terramycin molecule that accounts for the biological activity. These preliminary studies would suggest that the properties of the hydroaromatic molecule as a whole, rather than the presence or orientation of individual groups or, for that matter, of specific ring systems, determine its full potency. In addition, it would seem from these studies that certain of the peripheral groups are not essential for antibiotic activity.

Microorganisms have provided two of the most potent analogs of structure (XVII) which is common to terramycin and aureomycin for which we have proposed the name, tetracycline.* Were it not for the frequent obtuseness of nature, one might predict that this theoretical substance would have a high order of biological activity, since aureomycin lacks the C_5 hydroxyl in terramycin but, instead, possesses a chlorine group in position 7. This would suggest that the presence of the C_5 hydroxyl in terramycin or the C_7 chlorine in aureomycin in these positions were not absolutely essential to biological activity. Aureomycin and terramycin can be modified by elimination of the C_6 hydroxyl to yield anhydro compounds, which retain biological activity, despite aromatization of ring C. More radically modified systems, such as are produced in the apoterramycins, show no activity against gram-positive and gram-negative bacteria. After testing derivatives containing each ring system as it exists in terramycin, it would appear that none of the individual ring systems by themselves carries any distinguishable biological activity. Moreover, even in cases where the naph-

* Terramycin has, therefore, been assigned the generic name oxytetracycline.

thacene skeleton is preserved, there are limiting factors controlling the contribution of peripheral groups and the degree of aromatization on antibiotic action.

While this work is only of a preliminary nature, it is certain that any rewarding study of the mode of action of terramycin and aureomycin will have to embrace the contributions of active and inactive, closely related structural analogs to any of the metabolic processes involving bacterial cells.

Summary

In augmenting studies on the mode of action of terramycin, consideration is given to the effects of modifying the structure on biological activity and to the significance of individual chemical groups in the molecule. As a result of structure determinations, a host of degradation products have become available, which are being examined for antimicrobial action. From preliminary studies it has been found that relatively few modifications can be made in terramycin resulting in retention of biological activity. As yet, no substance has been found possessing biological activity that does not contain the tetracyclic hydroaromatic ring system in terramycin, such as is illustrated by the active derivatives, anhydroterramycin and desdimethyldesoxyterramycin.

It is certain that any fruitful study of the mode of action of terramycin must take cognizance of closely related active and inactive structural analogs.

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CHLORAMPHENICOL*

By D. W. WOOLLEY†

The structure of chloromycetin (chloramphenicol) differs in only four respects from that of the biologically essential metabolite phenylalanine. Experiments will be described that show that the antibacterial action of chloromycetin can be antagonized by phenylalanine. This antagonism can be demonstrated only over a narrow range of concentration of the drug, and only with minimally effective doses of chloromycetin. The antagonism is noncompetitive. The biological properties of all of the compounds intermediate between phenylalanine and chloromycetin will be described in an effort to throw light on the question as to whether or not chloromycetin may act on bacteria, partly because it interferes with the incorporation of

* This paper was presented as part of the Symposium on Chemical Structure of Antibiotics in Relation to Mode of Action at the Meeting of the Section of Biology on October 13, 1952.
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phenylalanine into a compound necessary for multiplication of the microorganisms. The findings of Gale that chloromycetin inhibits the net synthesis of protein in bacterial cells will also be discussed. Other recent findings that shed light on the mode of action of chloromycetin will be mentioned.

*SECTION OF MATHEMATICS AND ENGINEERING**

ENGINEERING PROBLEMS IN THE RECONSTRUCTION OF THE BROOKLYN BRIDGE†

By D. B. STEINMAN‡

Introduction

When the Brooklyn Bridge was built in 1883, the present-day demands for accommodation of vehicular traffic were beyond anticipation. At that time, the immediate necessity was to provide for mass transportation by means of rapid transit. The original cross-section of the bridge, shown in FIGURE 1, was well conceived for this purpose. The tracks for the trains were located between pairs of deep stiffening trusses. The inner trusses, adjacent to the inner rails, distributed the train loads along the inner cables. The intermediate trusses, adjacent to the outer rails, distributed the train loads among the transverse floorbeams which, in turn, carried the loads to the outer shallow trusses. The intermediate trusses, furthermore, acted as effective protective barriers between the trains and the vehicular traffic.

To keep pace with the rapidly increasing population of Brooklyn, the original two-car trains were soon lengthened to four and five cars. In 1898, additional transit facilities were provided in the form of trolleys, located adjacent to the intermediate trusses.

With the building of subways under the East River, patronage of the transit facilities dwindled to such an extent that the rapid transit trains were discontinued in 1944. The trolleys were routed over the train tracks, leaving the roadways for the sole use of vehicular traffic, as they were originally. The roadways, however, were only 16 ft. 7½ in. wide, which was uncomfortably narrow for modern traffic. With further decrease in the patronage of the trolleys, the City became saddled with a bridge that was not needed for rapid transit and was inadequate for vehicular traffic. Taking its cue from the trend to replace trolleys with buses, the City decided to eliminate the trolleys from the bridge and rebuild it to accommodate vehicular traffic only.

By removing the intermediate trusses, it was possible to provide two thirty-foot roadways, each wide enough for three ten-foot traffic lanes. The new cross-section is shown in FIGURE 2. A photograph of the completed new roadway is shown in FIGURE 3. A perspective of the reconstructed bridge is shown in FIGURE 4.

The problem of converting the general recommendations into practical plans and specifications for the actual work was left to the engineer. All suggestions were studied carefully. The reasons and analyses that prompted their adoption or omission provide examples in practical engineering. They are given in the following text.

Description of Bridge

The elevation in FIGURE 5 shows the unique physical features of the Brooklyn Bridge. The bridge is of the suspension type, with four main cables which are cradled (hanging in slightly inclined planes). The stiffening trusses pass through the towers where they are rigidly fixed against all motion. They are pin-connected to the anchorages which are located at the ends of the land spans. The bridge is "five-hinged," counting the pin-connection at each anchorage and a slip joint at each mid-span. The expansion or slip joints are located in the stiffening trusses at the centers of the land and river spans. This arrangement with slip joints at mid-span was necessitated by the incorporation of the system of diagonal stays radiating from each tower top to points of attachment in the bottom chord. The slip joints cannot transmit bending moment, but do transmit shear by means of tongues projecting across each slip joint from the top and bottom chords on one side of the slip joint to the chords on the other side.

* The Section of Psychology held a meeting on October 20 at which a paper, entitled A New Personality Test: The Graphomotor Projection Technique, was read by Samuel B. Kutash. The Section of Anthropology held a meeting on October 27 at which a paper, entitled Brachycephaly and Structure of Cranial Bones, was read by Franjo Ivanicek. The Section of Oceanography and Meteorology held a meeting on October 28 at which a paper, entitled Upper-Atmosphere Research by Means of Rockets and Meteors, was delivered by Fred. L. Whipple. These papers will be printed in a later issue.

† This paper, illustrated with lantern slides, was presented at a Meeting of the Section on October 17, 1952.

‡ Consulting Engineer, New York.

The stiffening trusses hang from the cables by wire-rope suspenders at 7 ft. 6 in. spacing, which is the general spacing of the floorbeams. In addition to these suspenders, the portions of the stiffening trusses adjacent to the towers are carried by the diagonal rope stays that originate at the tower tops and fan out to connect to the bottom chords of the stiffening trusses at 15-ft. spaces. In the region of the diagonal stays, since the stays partially relieve the vertical suspenders in carrying the dead and live load of the bridge, the curves of the main cables are flatter than where the loads are carried solely by vertical suspenders.

There are 125 panels in each 930-foot land span. The slip joint is at Panel Point 60. Panel Point 122 is at the anchorage. The diagonal stays are attached to the bottom chords of the stiffening trusses at alternate panel points, between Panel Points 7 and 55.

The main or river span is 1595.5 feet. There are 107 panels between each tower and the center of the river span where the slip joint is located. Symmetrical about each tower, the diagonal stays are attached to the bottom chords of the stiffening trusses at alternate panel points between Panel Points 7 and 55.

The stiffening trusses are "quadruple intersection Warren trusses." A vertical section anywhere between the verticals at the panel points will cut *four* eyebar web members. The members that slope downward toward the river are termed "diagonals," and are fixed in length. The members that slope upward toward the river are termed "counters," and are adjustable by means of sleeve nuts.

The entire system of cables, diagonal stays, and stiffening trusses is highly "redundant" or "indeterminate," rendering accurate stress analysis highly difficult and involved. A change in adjustment of any member produces a readjustment of stresses in other members and affects the vertical curvature and alignment.

A singularly unique condition exists on the Brooklyn Bridge. The small steel rollers under the saddles on top of the towers of the Brooklyn Bridge have never functioned as intended. The rollers simply have not rolled. They have become skewed, imbedded, and rusted, virtually "frozen" from the start. As a result, unbalanced cable-pull on the tower-tops, from unsymmetrical loading or thermal expansion of the cables, has produced serious bending strains in the towers, tending to loosen joints in the masonry (That is why the later suspension bridges have been built with flexible steel towers, to reduce and resist the bending stresses). In the Brooklyn Bridge, the cumulative unbalanced pull on the tower-tops has repeatedly caused the cables to overcome their friction and to "jump" in their saddles, thereby alarming the public but, actually, producing a readjustment of strain and relief of stresses. In 1922, two of the main cables on the Manhattan tower slipped in the saddles and moved toward the river by ½ and 1¾ inches, respectively, thereby giving evidence of excessive unbalanced cable pull.

The unbalanced horizontal force on the tower tops of the Brooklyn Bridge combined with the vertical load produces a resultant that comes to the edge of the critical "core-area" of the horizontal masonry joints of the tower (corresponding to the "middle-third" in simpler sections). Any aggravation of this condition, as by adding unbalanced dead load to main span and side spans, might be critical.

In the case of the Brooklyn Bridge towers, there is a very fortunate saving feature. The towers rest on compressible timber cushions formed by the roofs of the caissons on which they are founded. These massive timber cushions are fifteen feet thick under the Brooklyn tower and twenty-two feet thick under the New York tower. The elastic compressibility of the timber cushion permits the masonry tower to rock on its base, thereby relieving the strains in the masonry from unbalanced cable pull. Precision surveys with transit show that the deflection graph for each tower is a straight line, as in a hinged column, and not a curve as in a cantilever beam. This action has undoubtedly saved the tower masonry from serious racking strains in the past.

The effect of unbalanced cable pulls on the towers was of constant concern during the work of reconstruction. It governed the erection procedure and even affected the choice of type of floor slab.

Features of Reconstruction

At the time the Brooklyn Bridge was planned and built, the science of suspension-bridge design was still in its crude infancy. It is a credit to the intuitive genius of the builder that the structure has endured. Nevertheless, a number of features of the design are seen to be unscientific, in the light of modern theory and knowledge. The reconstruction of the bridge afforded an opportunity

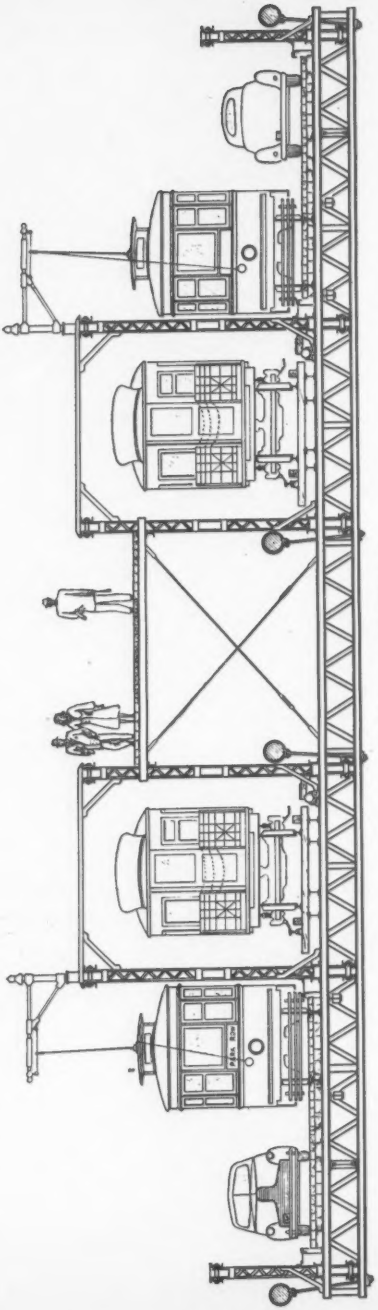


FIGURE 1. Cross-section 1898 to 1944.

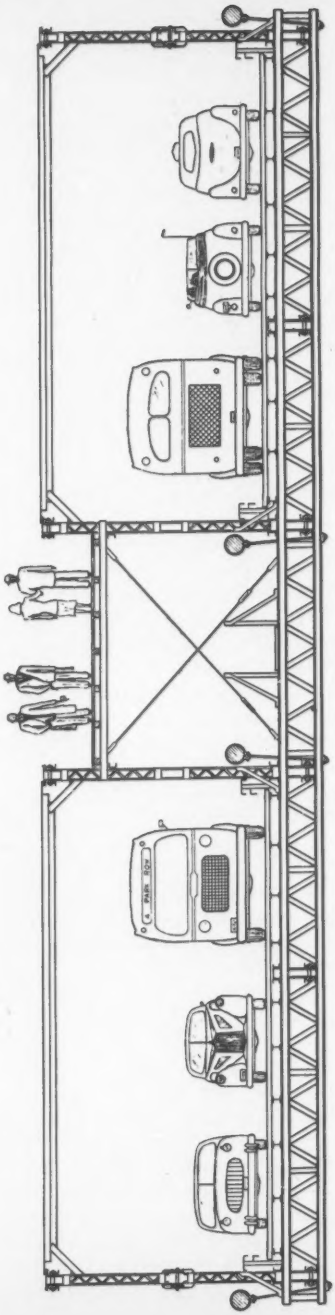


FIGURE 2. Reconstructed cross-section.

and provided the necessity of making some essential changes in the basic design, in order to secure the desired improved efficiency and enlarged capacity.

The primary and outstanding change that was necessary was the simplification and rearrangement of the stiffening trusses. The Brooklyn Bridge was built with *four* cables and *six* stiffening trusses—two inner trusses, 17 feet deep; two intermediate trusses, 17 feet deep; and two outer trusses, only 8 feet 9 inches deep. The inner and outer trusses were located under the four cables, but the two intermediate trusses had no cables above them to which they could transmit their share of the total load. This was an unscientific arrangement, materially impairing the efficient functioning of the structure as a whole. Moreover, the intermediate trusses interfered with the desired clear roadway width of 30 feet, leaving a roadway width of only 16 feet 7.5 inches. This was too narrow for two lanes of modern highway traffic. Cars could barely squeeze two abreast through such restricted width and, in actual use, it was virtually only a single-lane roadway. Both necessity and efficiency required, therefore, the removal of the two intermediate trusses, thereby simplifying the cross-section by reducing the number of stiffening trusses from six to four, one under each cable and, at the same time, yielding two clear roadways, each 30 feet wide, or a total of six highway lanes for vehicular traffic.

This scientific rearrangement of the cross-section also called for the deepening of the two outer trusses from their half-depth of 8 feet 9 inches to the same full depth, 17 feet, of the other trusses, so that the outer trusses could take their proper share of the total load and transmit that share effectively to the cables above them. With their half-depth, the outer trusses were not doing their share of the work. They were contributing only 10 per cent to the total vertical stiffening of the bridge. With their reconstruction to full depth, they are now contributing 50 per cent of the total vertical stiffening of the bridge.

The combined effect of the change was equivalent to removing the full-depth intermediate trusses from their ineffective and objectionable intermediate location and transferring them to replace the inadequate half-depth trusses under the outer cables. Actually, in the interests of economy of labor and material, the half-depth outer trusses were left in place but were built up to the full-depth uniform truss-height by utilizing salvaged top chords from the scrapped intermediate trusses.

The new eyebars that were placed in the outer trusses were all carefully adjusted with the aid of precision strain-gauge measurements.

As indicated in the cross-sections, both in the original design and after reconstruction, each stiffening truss has an intermediate chord, consisting of one or two longitudinal 6-inch channels, sitting on top of the floorbeams and riveted to the vertical posts of the stiffening trusses. In removing the intermediate trusses, the bottom chords were left in place for participation in resisting the horizontal thrust from the diagonal stays; and the intermediate chords were also left in place to form the top chord of a new distributing truss to aid the floorbeams. The intermediate trusses were removed by flame-cutting the diagonals and counters close to the pins, and flame cutting the vertical posts at the tops of the intermediate chords.

The intermediate trusses, although they were not directly suspended from any cables, performed the valuable function of distributing the effect of concentrated floor loads. With the intermediate trusses completely removed, each floorbeam would have to carry its wheel loads without help from adjacent floorbeams. This would have overstressed the floorbeams. To improve this condition, light diagonal members were added between the retained bottom chords and intermediate chords. In this manner, stiff *distributing trusses* were formed that reduce the stresses in the floorbeams due to concentrated wheel loads by fifty per cent.

Other incidental features of the reconstruction work, such as choice of type of floor slab and stringers, reinforcement of the bottom chords, and methods of procedure are described under their respective headings.

Capacity of the Bridge

The live load originally proposed for the reconstructed bridge was 40 lb. per sq. ft. of roadways or 2400 lb. per lin. ft. of bridge, predicted on an unchanged value of the dead load.

Actually, the reconstruction of the bridge increased its weight by 400 lb. per lin. ft., which is about 7 lb. per sq. ft. of roadway. Allowing 8 more lb. per sq. ft. for unforeseen additions, there is left an allowable live load of 25 lb. per sq. ft. This is an ample figure when it is considered that

FIGURE 3. New roadway.

traffic on the bridge will be restricted to passenger automobiles.

The value of 25 lb. per sq. ft. applies to load over the entire bridge, affecting main carrying members, such as towers, cables, anchorages, and stiffening trusses. The local carrying members such as floor slabs, stringers, floorbeams, and suspenders, are strong enough to carry trucks and to permit the passage of properly spaced maintenance vehicles, fire trucks, and other emergency vehicles. During construction operations, trucks weighing over 20 tons frequently traversed the bridge over the new floor.

A comparison between the average dead loads of the bridge, in pounds per linear foot, before and after reconstruction, is given in the following table

	Before reconstruction	*After reconstruction*
For each outer cable	2,220	2,400
For each inner cable	2,500	2,520
Total for bridge	9,440	9,840

The total dead load of the bridge has been increased by about 4 per cent, but the division of dead load between the inner and outer cables has been more nearly equalized. The maximum dead load per cable, taken by the inner cables, has been increased less than one per cent.

Rebuilding the Outer Trusses

It was feared that, if the outer trusses were to be unpinned, variations in the adjustment of the suspenders and diagonal stays would then be free to distort the deck. To avoid such unpredictable complications, the method adopted for building up the outer trusses was one that did not require dismantling of the existing trusses.

The details of the built-up outer trusses are shown in FIGURE 6. Holes were cut in the cover plates of the old top chords to permit the insertion of new gusset plates, recessed to avoid interference with the old posts. New diagonals and counters and extensions of the posts were attached to the

FIGURE 4. Perspective of reconstructed bridge.

new gusset plates. The extensions of the posts were spliced to the old posts by 7-inch channels that straddled the old chords and were bored to take the old top chord pins. The top chords of the built-up trusses were salvaged from the dismantled intermediate trusses. It was required to fabricate special new top chord sections at the towers, because interference from the masonry prevented re-use of the old sections. The new diagonals and counters were made adjustable to accommodate them to variations in dimensions of the old structure.

New Roadway Slab

The Brooklyn Bridge roadways were originally of light construction, consisting of timber planks. These were later replaced with wooden paving blocks. This original light-weight construction limited the permissible dead load of any new construction. The use of a conventional modern reinforced concrete slab for the new roadways was precluded.

The official authorities recommended an open steel grid for the new roadways, so as not to increase the dead load of the bridge. This was favored by the Maintenance Department, as the open grating would eliminate snow removal and sanding. There was one difficulty, however. While asking for an open grating for the roadways in the main span over the river, the officials insisted upon a solid floor construction in the flanking land spans. More than half the area under the two land spans comprises streets, sidewalks, open docks, and uncovered industrialized space. The authorities feared that this area would be menaced by objects falling through the open grating.

The proposed combination of open grating in the river span with solid slab in the land spans was, however, impracticable, for the unbalanced dead load in main span and side spans would produce a serious unbalanced cable pull on the towers.

The adoption of a uniform roadway construction for the three spans was therefore recommended.

The use of an open grating in all three spans with

a net under the land spans was considered and discarded. Another idea considered a closed floor in the land spans and an open grating in the river span, counterweighted with heavy concrete curbs to equalize the weights, but this would have been uneconomical.

It was finally decided to use a closed floor of the lightest possible type. The new roadway slab is of 3-inch "I-Beam-Lok," with a special arched pan under each square of the mesh to reduce the volume of concrete. The widest spacing of stringers is only 3 ft. 6 in., which makes the I-Beam-Lok strong enough, even without the concrete filling, to carry trucks weighing more than 20 tons. The new roadway slab with the concrete filling, was instrumental in increasing the dead load of the bridge.

To expedite reconstruction, a construction joint was placed in the floor slab along the center stringer of each roadway. The Contractor was thus able to work from the old roadway and complete all demolition and construction adjacent to the inner trusses. Then, working from the new half-roadways, he completed demolition and construction adjacent to the outer trusses.

New Roadway Stringers

The reconstruction of the roadways required a complete new set of longitudinal stringers.

The new roadway stringers are 8-inch 23-pound standard I-beams. Their span is short, the floor beams being spaced 7 ft. 6 in. They were chosen (1) to provide the standard headroom of 14 ft. 0 in. for vehicular traffic, and (2) to have strength consistent with that of the floor slab. The new stringers can carry 18-ton trucks in a single lane and 16-ton trucks in multiple lanes without overstress.

The depth of the new stringers is the same as that of the old stringers under the train tracks, and it was hoped that the track stringers could be used under the new roadway. It was found, however, that the corrosion of the top flanges under the track ties was far advanced, and the idea of re-using the old track stringers had to be abandoned.

The new stringers were furnished in 30-foot lengths and were erected with staggered joints. In the regions of the diagonal stays, the stringers and floor slabs have no expansion joints. This permits the floor to participate in resistance to thrust from the diagonal stays and also provides lateral stiffening for the unbraced bottom chords. Outside the regions of the diagonal stays, the stringers and floor slab are provided with expansion joints at 90-foot intervals.

Floorbeams and Distributing Trusses

In the reconstructed cross-section of the bridge, the floorbeams were found only strong enough to carry three 7.2-ton trucks abreast without overstress. This indicated the necessity for reinforcing the floorbeams to make their capacity consistent with that of the stringers.

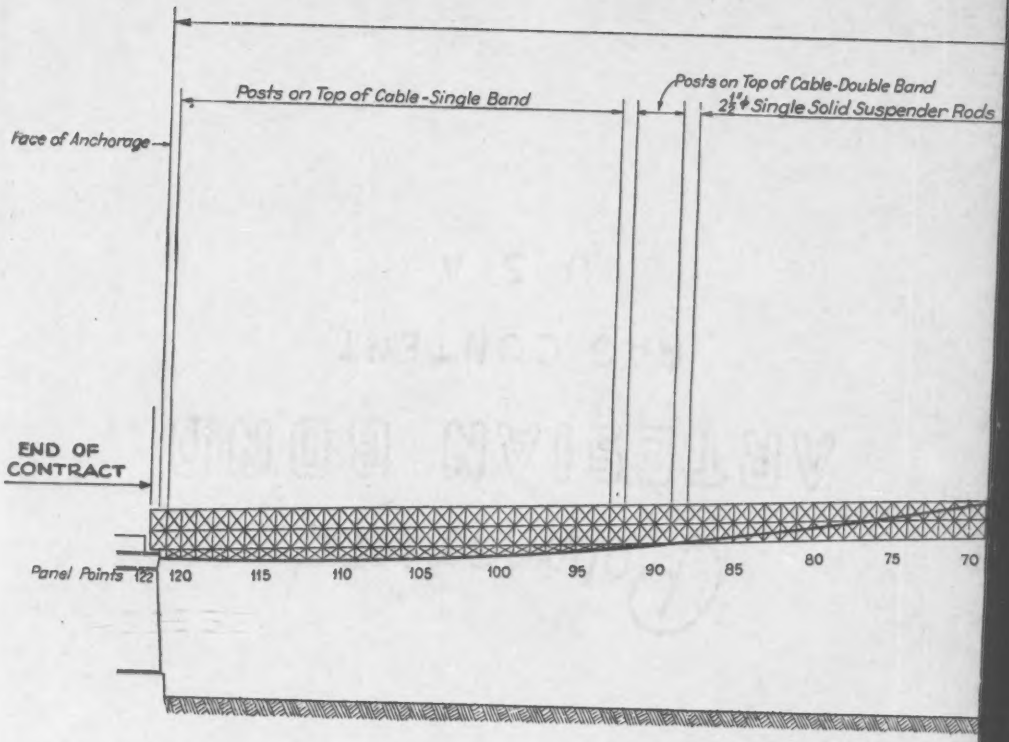
Reinforcement of the floorbeams would have been very difficult and, fortunately, was made unnecessary by converting the remnants of the old intermediate trusses into distributing trusses. The function of the distributing trusses is to force adjacent floorbeams to carry part of the load that is concentrated on any single floorbeam. By this expedient, the capacity of the floorbeams was doubled, permitting them to carry three 15-ton trucks abreast.

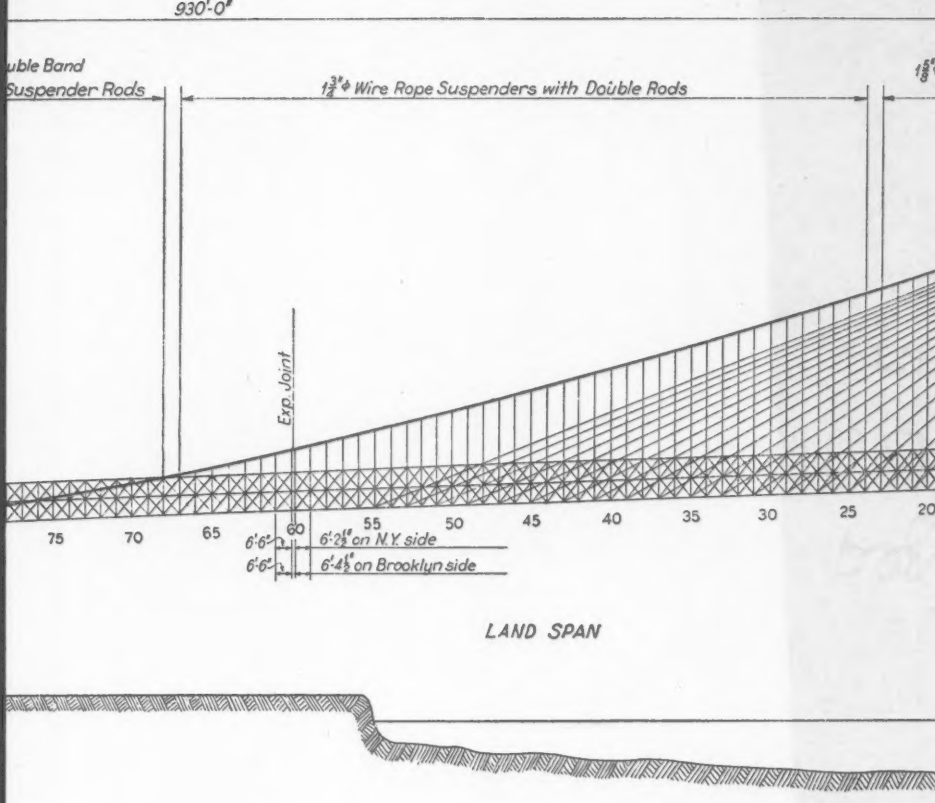
Comparison of FIGURES 1 and 2 shows that the entire intermediate trusses were not removed. The bottom chord and the intermediate chord, which consists of two 6-inch channels, were left in place. The addition of light diagonal members converted the two chords into effective trusses. Where the bottom chord is of double depth, between Panel Point 17 in the land span and Panel Point 17 in the river span, the two chords were joined by solid web plates to form distributing girders.

The calculations for obtaining the stresses in the interacting floorbeams and stiffening girders were not specifically related to suspension bridges but formed an interesting problem in the analysis of structures of multiple redundancy. The arrangement and loading of the floorbeams and distributing trusses are shown in FIGURE 7. To simplify the computations for determining the stresses in the system, the distributing trusses were assumed to extend four panels. The effect of this simplification on the calculated stress in the center floorbeam, which carries the heavy rear truck wheels, is small and on the safe side.

The stringers that sit on the top flanges of the floorbeams also aid in distributing loads but, because of lack of continuity, their effect is variable.

The method of computing stresses in the floorbeams and distributing trusses is based on the relations of the deflections of the members. The fundamental deflection equations for the arrangement of members in the floorbeam system are


Posts on Top of Cable - Single Band
Posts on Top of Cable-Double Band
2½" ϕ Single Solid Suspender Rods
Face of Anchorage
END OF CONTRACT
Panel Points 122 120 115 110 105 100 95 90 85 80 75 70


930'-0"
uble Band
Suspender Rods
1¾" Wire Rope Suspenders with Double Rods
Exp. Joint
75
70
65
60
55
50
45
40
35
30
25
20
6'-6"
6'-6"
6'-2½" on N.Y. side
6'-4½" on Brooklyn side
LAND SPAN

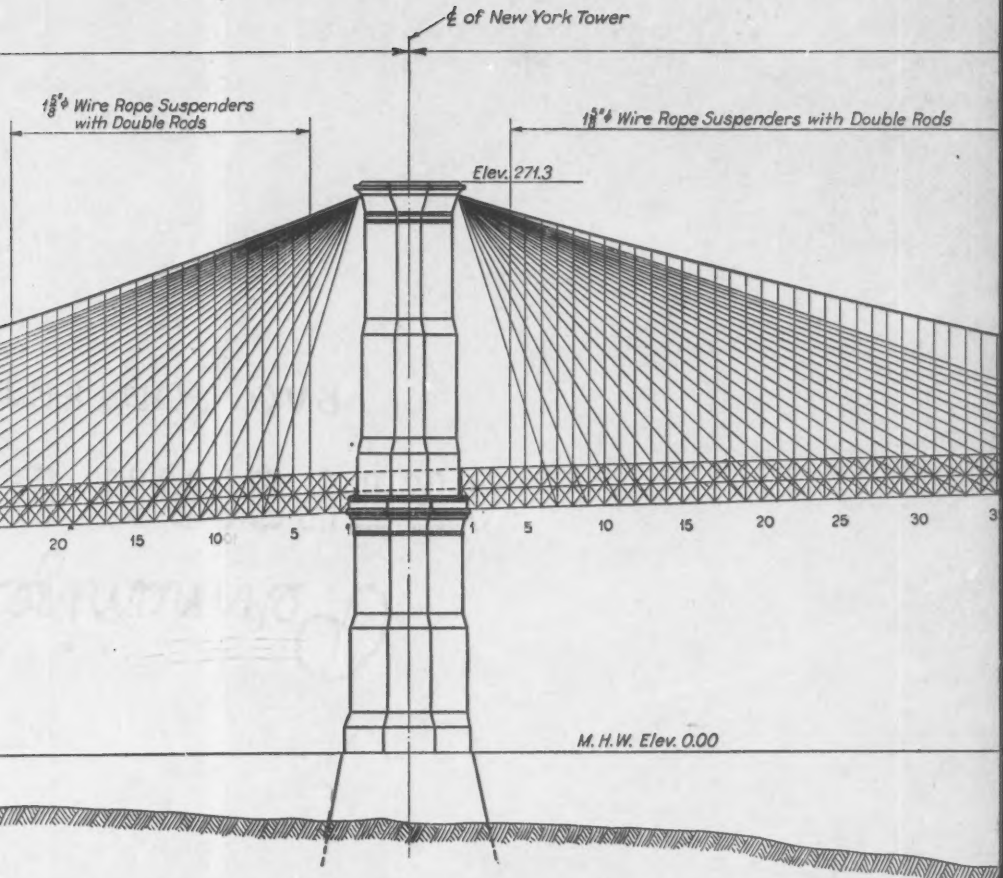


FIG. 5 HALF ELEVATION OF BRIDGE

0 50 100 150 200 FT.

SCALE

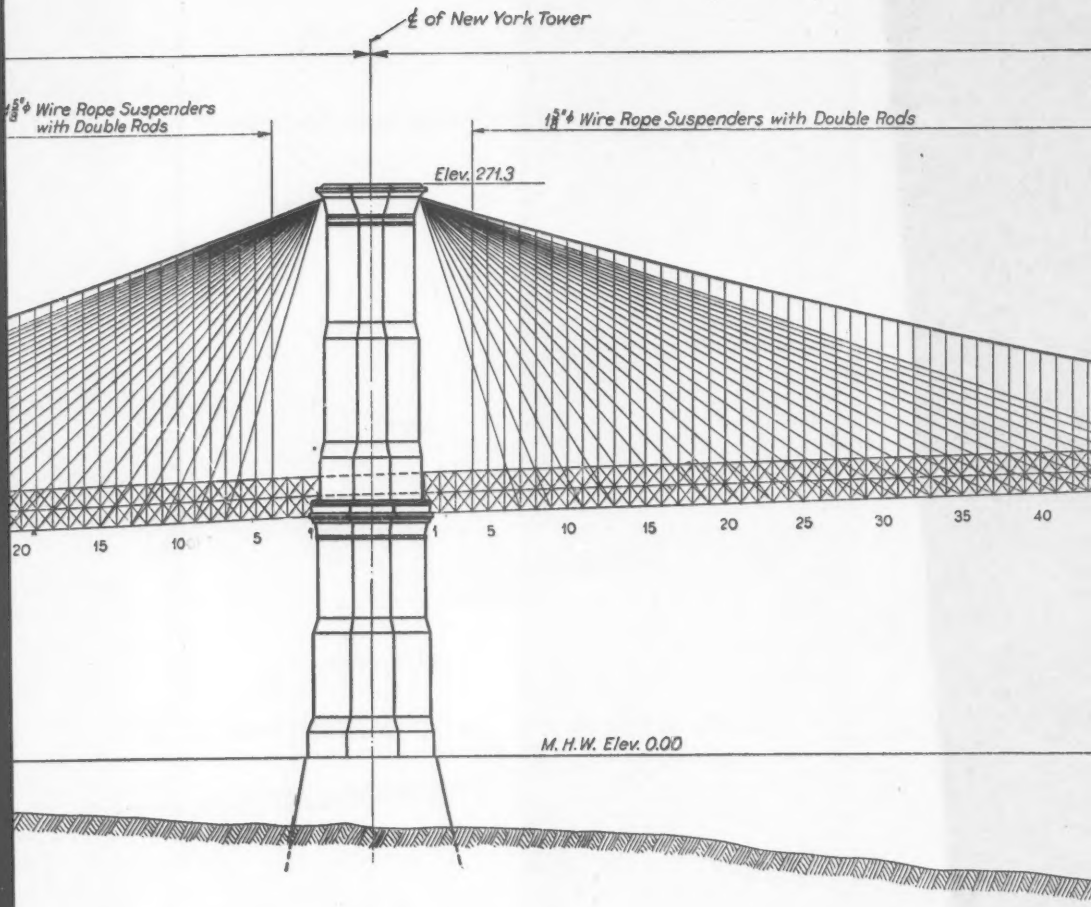
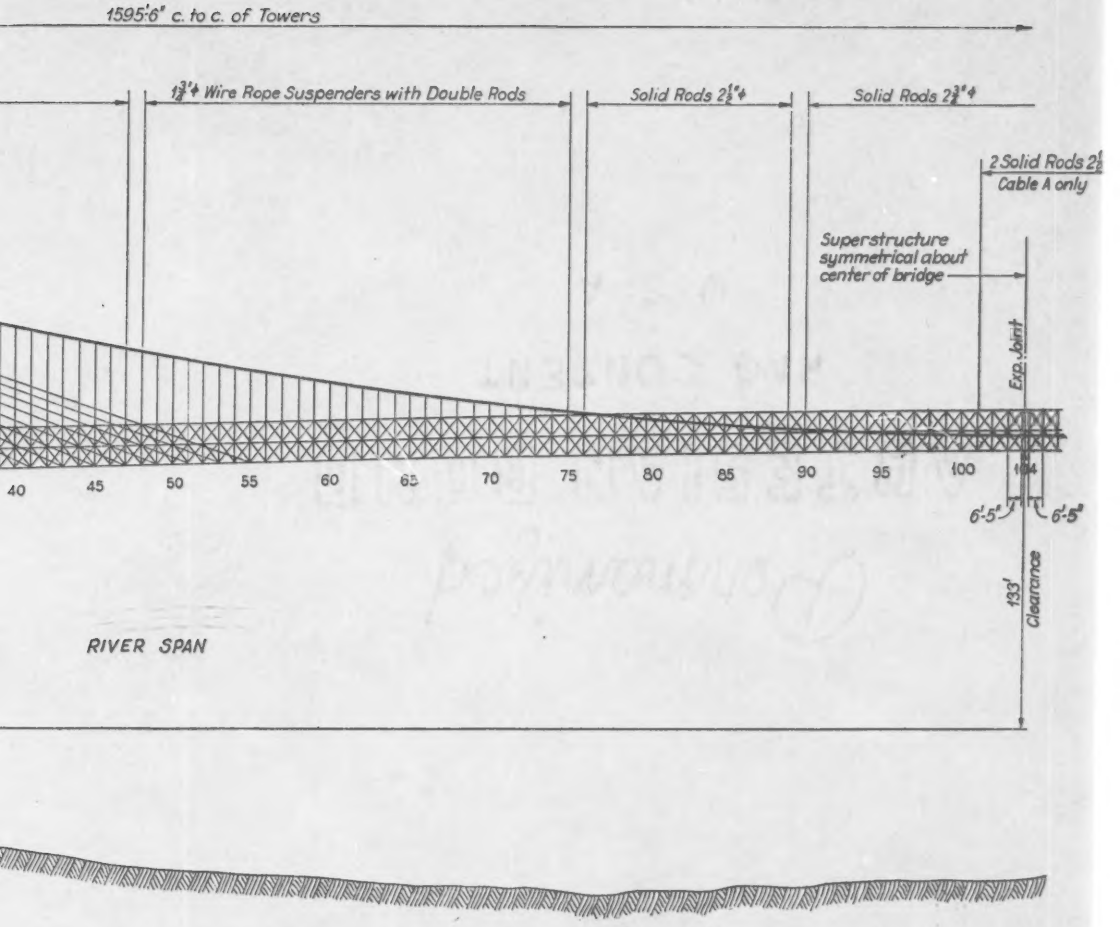


FIG. 5 HALF ELEVATION OF BRIDGE

0 50 100 150 200 FT.

SCALE


1595'6" c. to c. of Towers
1 3/4" Wire Rope Suspenders with Double Rods
Solid Rods 2 1/2"
Solid Rods 2 3/4"
2 Solid Rods 2 1/2"
Cable A only
Superstructure
symmetrical about
center of bridge
Exp. Joint
40
45
50
55
60
65
70
75
80
85
90
95
100
104
6'-5"
6'-5"
133'
Clearance
RIVER SPAN

given in FIGURE 8. After substitution of numerical values, solution of the equations gives the reactions between the floorbeams and the distributing truss from which stresses are computed. The reduction of stress in the floorbeams is apparent from the deflection curves shown in FIGURE 8 with and without consideration of the distributing truss.

The distributing trusses are, however, not adequately effective in reducing stresses in the end floorbeams at the slip joints. To give further support to these end floorbeams, auxiliary distributing trusses, three panels long, were built, 6 ft. 9 in. away from and parallel to the main distributing trusses. In this way, the end floorbeams at the slip joints are braced and stiffened by two intermediate distributing trusses instead of one. A computation similar to that shown in FIGURES 7 and 8 was made for this case. This computation was considerably more involved, as the arrangement of members is unsymmetrical and as there are two distributing trusses.

Calculation of Stresses in the Cables and Stiffening Trusses

The cables and stiffening trusses of the Brooklyn Bridge form a highly redundant structure. The degree of redundancy may be visualized by starting with the simplest components of the bridge and then adding the parts required to build the bridge up to its full form.

Basically, the bridge is formed of trusses fixed rigidly to the towers and cantilevering half way into the land and river spans. In each land span, a simple truss is placed between the end of the cantilever and the anchorage. In the river span, the two cantilevers are pinned together at midspan, forming the first redundancy. Then the cables and suspenders are added, forming the second redundancy. Finally, the diagonal stays are added, each stay adding a redundancy. To simplify the effect of the stays, they were combined into three groups at each side of the towers.

Thus, each land span had four redundancies, and the main span had eight redundancies.

The equations involving these redundancies did not yield the exact stresses, as they took no account of the distortion of the structure, which alters the computed stresses. In lieu of determining the effect of distortion by extended mathematical treatment, deflection and stress measurements were made on the bridge under train loads and compared with the values computed from the equations. The experimental data thus obtained provided checks on the computations and furnished correction factors to apply to the computations to account for distortion of the bridge under load.

The work described above had to be checked carefully to withdraw the data required for application to the conditions of loading imposed by the removal and addition of material during the various stages of reconstruction of the bridge. The end results are presented herein.

Reinforcement of the Bottom Chords

A careful analysis of the stresses in the stiffening trusses revealed high compressive unit stresses in the bottom chords in the regions of the diagonal stays. These high stresses were produced by the superposition of thrust reaction from the diagonal stays added to the stresses from normal truss action. The maximum unit stresses in the bottom chord, before reinforcement, are shown in FIGURE 9b. It was decided to reinforce the bottom chords in the overstressed regions where the calculated unit stresses exceeded 30,000 pounds per square inch. The regions in which the bottom chords required reinforcement extended between Panel Points 17 and 45 in the land spans and between Panel Points 17 and 42 in the river span.

The existing bottom chords between Panel Point 17 in the land span and Panel Point 17 in the river span are of double depth, each half-section consisting of a 9-inch channel set on top of another 9-inch channel with webs vertical. The regions requiring reinforcement were beyond the double-depth chords, calling for lengthening the extent of the double-depth chords. Instead of using 9-inch channels to be set on top of the existing 9-inch channels, it was decided to use 8×4 angles for the new material. With suspenders and diagonal stays in the way, the angles were easier to erect than channels.

To make the bottom-chord reinforcement effective in sharing stress, it was applied when the stresses in the bottom chord were reduced to as low a value as controlled removal of the dead load of the original floor permitted.

Within the main towers, the bottom chords are in contact with the tower masonry on one side, and close to masonry ledges on the underside. Pockets thus formed were holding dirt and moisture and were causing considerable corrosion of the lower flanges of the bottom chords. Cramped quarters made general reinforcement by riveting impracticable, and the compression stress in the bottom chord precluded use of welding.

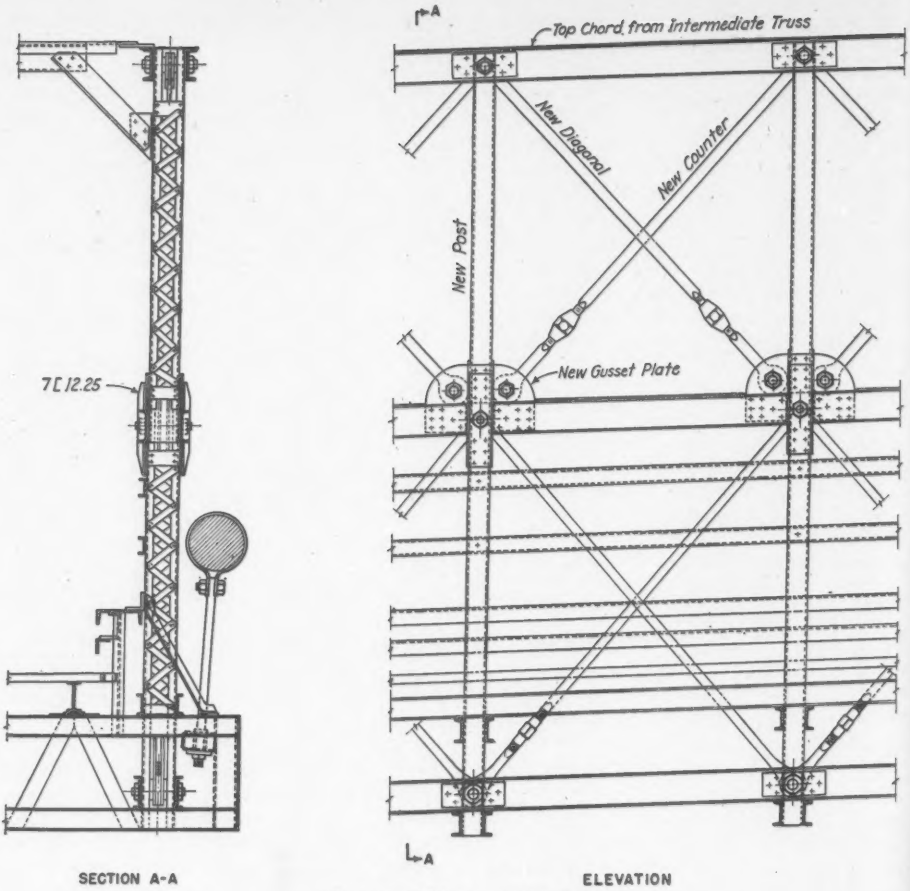


FIGURE 6. Addition to outer trusses.

At the faces of the towers, where cutbacks in the tower masonry provided room for riveting, the lower flanges of the bottom chords were reinforced locally to rest on new bearings. Between the bearings, the lower chords were cleaned thoroughly and encased in concrete. The concrete was carried up high enough to serve as curbs for the roadways. In this way, the chords are stiffened to increase their load-carrying capacity, while they are also protected from dirt and moisture.

New Top Struts

The connections and bracing that held the original shallow outer trusses upright were considered inadequate when the outer trusses were raised to the height of the inner trusses. The flexibility of the elongated web members rendered the new top chords liable to lateral buckling under the compressive stresses imposed by live load. To eliminate this hazard, the new top chords were tied by means of stiff struts to the top chords of the inner trusses which, in turn, are braced by the promenade construction.

The new top struts consist of two channels to form an efficient T-section. The vertical channel provides vertical stiffness. The horizontal channel provides lateral stiffness for the compression flange. Knee braces at the trusses provide open spaces, to facilitate any future removal of pins from the top chords. Another advantage of the T-section is in its flat top, which does not hold debris.

At the towers, special sections were required for

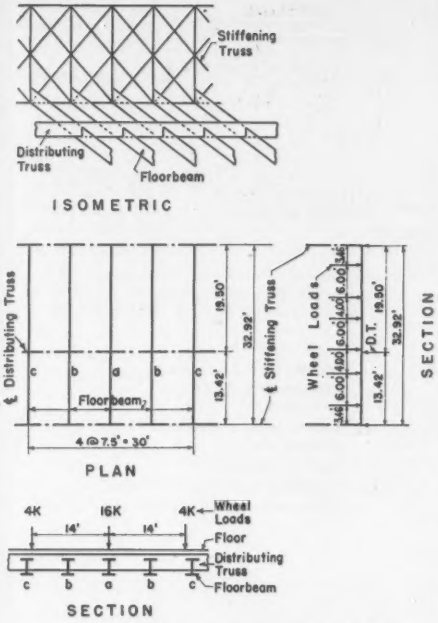


FIG. 7 FLOORBEAMS & DISTRIBUTING TRUSS

the struts to support the promenade where it divides to pass through the masonry arches at the level of the top chords.

Reconstruction of the Promenade

The timber promenade is the most hallowed feature of the Brooklyn Bridge. It has served and given pleasure to countless thousands through the years, including those walking to work across the span as well as those strolling for pleasure. At the same time, the promenade has been the greatest nuisance for maintenance, especially whenever the timber stringers under the floor planks started to decay. The timber promenade and its supporting stringers required complete replacement, and due consideration was given to the use of permanent and fireproof materials. All substitutes for timber, however, increased the dead load, and the dead-load problem had already been made acute by the adoption of a closed roadway slab. For this reason, as well as to preserve the tradition and comfort of the elevated boardwalk, it was decided to retain the timber flooring for the promenade.

The new promenade was laid to the boardwalk specifications of the Park Department. The stringers are creosoted for preservation, and the planks are salt treated for fire prevention. The opportunity was taken, after removal of the old promenade, to replace with steel all timber supports under the stringers.

Installation of an Inspection Walkway

The space under the promenade had been used to carry the electric cables for trolleys, trains, telephones, and lighting. These cables, with the exception of a 1,000,000 CM drainage cable for the protection of telephone cables against electrolysis, have all been removed, leaving the space clear. In order to provide convenient access, along the length of the bridge, for inspection and maintenance, an inspection walkway was installed in this space. This was laid on the floorbeams, with cross walks connecting to the roadways every 120 feet.

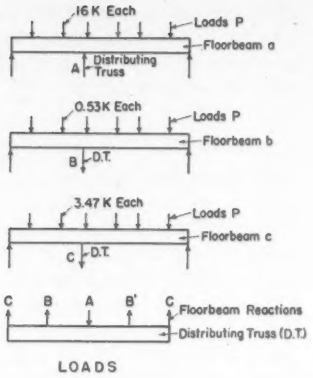
The inspection walk serves several purposes: It partially closes a dangerous open space; it provides a storage space for materials when repairs are to be made; it provides crossovers between the roadways; and it makes it unnecessary for maintenance men to walk long distances along the busy roadways.

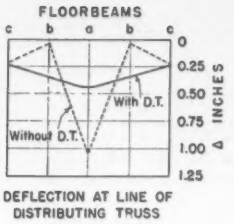
The inspection walk is a light open steel grating protected by wire rope strand handrails.

Riveting vs. *Welding*

At the request of the Department of Public Works, wishing to avoid the use of welding, the details in the contract drawings for the reconstruction of the bridge were made up entirely with riveted connections. This limitation was especially restricting, because of the inconvenient dimensions of the old steel channels of the bridge members, fabricated when the application of steel was in its infancy. Compared with modern channel sections, the old steel channels in the Brooklyn Bridge have thicker webs and narrower flanges. Rivets through the old channel flanges were necessarily small and difficult to drive.

Fastening of the new longitudinal stringers to the top chords of the old latticed floorbeams posed a problem. The top chords of the old floorbeams consist of two 6-inch channels, back to back, with flanges horizontal. It was impossible to drive rivets through the channel flanges, because of their narrowness and, in addition, the presence of web reinforcing plates between the channel flanges. Connection between the new stringers and the old floorbeams was accomplished by means of clip





NOTATION

Δ_A : Net Deflection of F.B. a at D.T.

δ : Deflection of F.B. at D.T. due to Unit Load at D.T.

δ_{AA} : Deflection of D.T. at A due to Unit Load at A.

A : Reaction of F.B. at A.

ΔP : Deflection at D.T. due to Wheel Loads.

DEFLECTION EQUATIONS

Floorbeam a: $\Delta_A = \Delta P - A\delta$

Floorbeam b: $\Delta_B = \Delta P + B\delta$

Floorbeam c: $\Delta_C = \Delta P + C\delta$

Distributing Truss: $\Delta_A = \Delta_C + A\delta_{AA} - 2B\delta_{AB}$

Distributing Truss: $\Delta_B = \Delta_C - B\delta_{BB} + A\delta_{AB} - B\delta_{BB}$

Distributing Truss: $A = 2B + 2C$

FIG. 8 DEFLECTIONS OF FLOORBEAM SYSTEM

angles, riveted to the vertical webs of the channels that form the top chords of the old floorbeams.

When work started, it was found that drilling holes for rivets in the old floorbeams was a very slow and difficult process. It seemed that the old steel had hard spots that resisted and dulled the drills. Finally, to expedite the work, the Contractor's proposal to weld the new stringers to the old floorbeams was accepted. The clip angles were eliminated. The beveled plates, filler plates, and bearing plates required between the stringers and floorbeams were reduced in size.

The distributing truss, which was field riveted in the south roadway, was almost entirely field welded in the north roadway. With the exception of the work on the outer trusses and the reinforcement of the bottom chords of the four trusses, welding was substituted for riveting as the work progressed, with consequent saving in weight of material. More important, valuable time was saved in completing the work and opening the roadways to traffic.

Miscellaneous Studies

The reasons that prompted the omission of certain features of suggested reconstruction work are worthy of mention. They involved as much study and consideration as some of the main features of reconstruction incorporated in the work.

The vertical profile of the bridge roadways is far from ideal. There are dips at the towers that give the appearance of local settlement of the bridge trusses at these points. Actually, the trusses have not settled more than one inch at the towers since they were built. The dips are due to a humping up of the trusses in the regions of the diagonal stays. A search of records pertaining to the bridge failed to reveal the reason for these humps. If they were built into the structure originally, straightening of the humps would induce high stresses in the stiffening trusses, with possible hazardous results. It was decided, therefore, to leave the vertical profile uncorrected. Correction, moreover, would have involved the adjustment in length of over a thousand diagonal stays and vertical suspenders.

A similar problem was posed by the horizontal alignment of the bridge. In 1898, a traffic block piled up trucks and trolleys on the south roadway, and the trusses in the Brooklyn land span buckled laterally about 3 inches. This deflection has remained unchanged to this day. Consideration

FIG. 9a SCHEMATIC ELEVATION OF BRIDGE

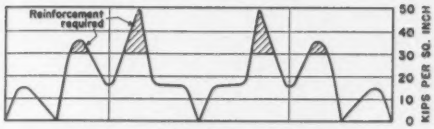


FIG. 9b MAXIMUM UNIT STRESSES
BOTTOM CHORD OF STIFFENING TRUSSES

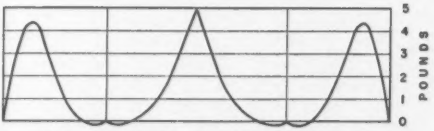


FIG. 9c INFLUENCE LINE FOR CABLE PULL

was given to correcting this defect, but study showed the proposed operation to be costly. As the deflection had not increased in 53 years and the new stiff floor would minimize future aggravation of the condition, the plan for correction of the horizontal alignment of the bridge was abandoned.

The tensile stresses in various counters of the stiffening trusses had been measured and were found high. Diagonals and counters had broken in the past from overstress during the period when the bridge carried rapid transit trains. These breakages, however, practically ceased when train traffic was suspended. It is likely that these breakages would cease entirely under the less-concentrated loading of vehicular traffic. The cost of putting gage points on the thousands of web members and performing adjustments in the face of changing temperature and loading was deemed to be far greater than the possible replacement of an occasional broken web member, and the suggestion for adjusting the diagonals and counters was not followed. Besides, the high stresses in the counters may have been induced deliberately to take the slack out of inaccurately fabricated diagonals, and lowering of the stresses in the counters might then produce the undesirable condition of loose diagonals.

Program of Demolition and Erection

With the main cable saddles frozen at the tower tops, the heavy masonry towers must act as anchorages to resist unbalanced cable pull that is caused by load and temperature.

Mindful of the objectionable effects of unbalanced cable pull, a program of procedure for demolition and erection was included in the specifications to maintain a balance of cable pulls in the land and river spans. The computations were based on the influence lines for cable pull in the respective spans (shown in FIGURE 9c). The ordinates to the influence lines show the cable pull produced by the addition or subtraction of one pound of load at any point on the bridge. The influence lines indicated that work had to proceed simultaneously at four points on the bridge, located symmetrically about the towers.

The south roadway of the suspended spans was reconstructed, with its stiffening trusses, while one-way traffic was maintained on the north roadway, Manhattan bound in the morning, Brooklyn bound in the afternoon and evening. The reconstructed south roadway was opened to traffic in May 1951, and the north roadway was then closed for reconstruction. The latter was completed in May 1952, but cannot be opened to traffic until reconstruction of the north roadway of the approaches is completed.

Contracts for the reconstruction of the approaches have been let to the same Contractor who reconstructed the suspended spans. This work is expected to be completed late in 1953.

Cost

The cost of reconstructing the suspended spans of the Brooklyn Bridge will be about $3,325,000.

Consideration was also given to replacement of the entire suspended structure, while retaining the present cables, towers, and anchorages, for an estimated cost of $8,000,000. The advantages from the extra expenditure would be increased life and unlimited load capacity. The latter advantage, however, could not be utilized without jamming the City Hall district with industrial traffic.

Rebuilding of the approaches to provide facilities to match the new roadways on the suspended spans is now under contract for about $3,600,000. The principle feature of the reconstruction of the Manhattan Approach was the restoration with reinforced concrete of 16 brick arches that had been cut to accommodate two train tracks. In the Brooklyn Approach, the south end, which swung westward, was demolished and swung to the east to connect to a newly formed express street. The new approaches are shown in FIGURES 10 and 11.

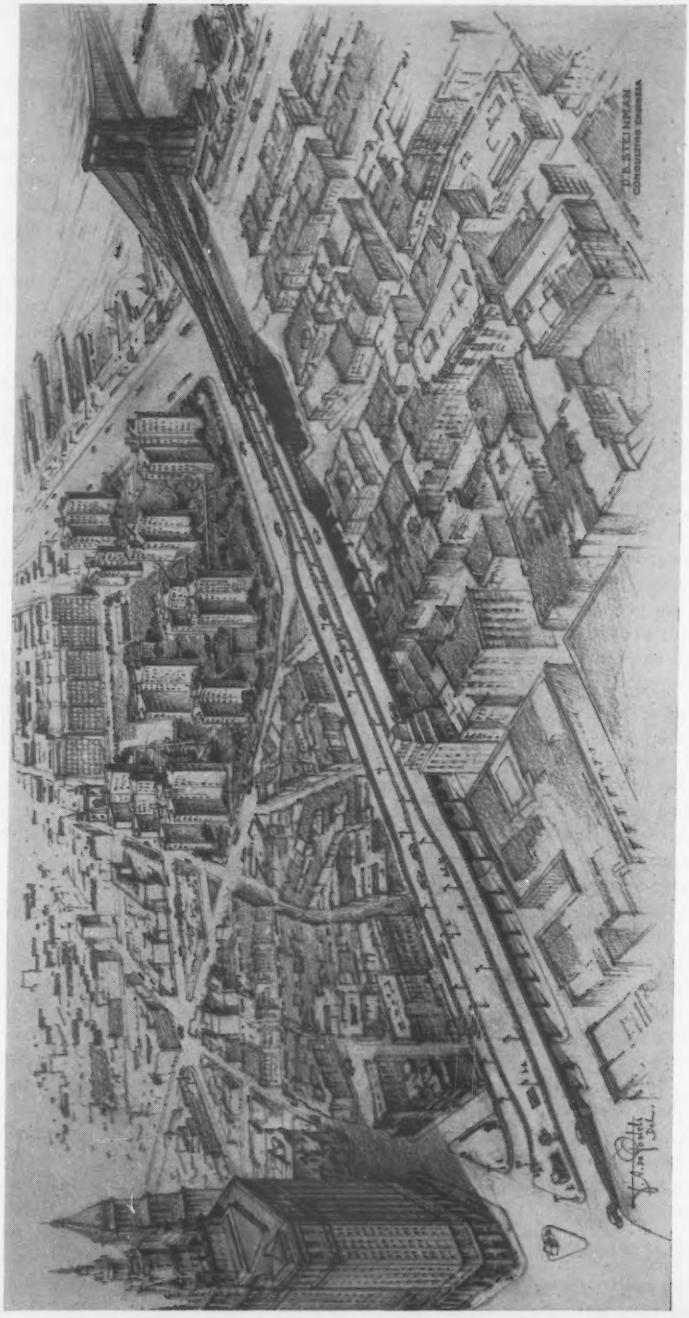


FIGURE 10. Rebuilt Manhattan approach.

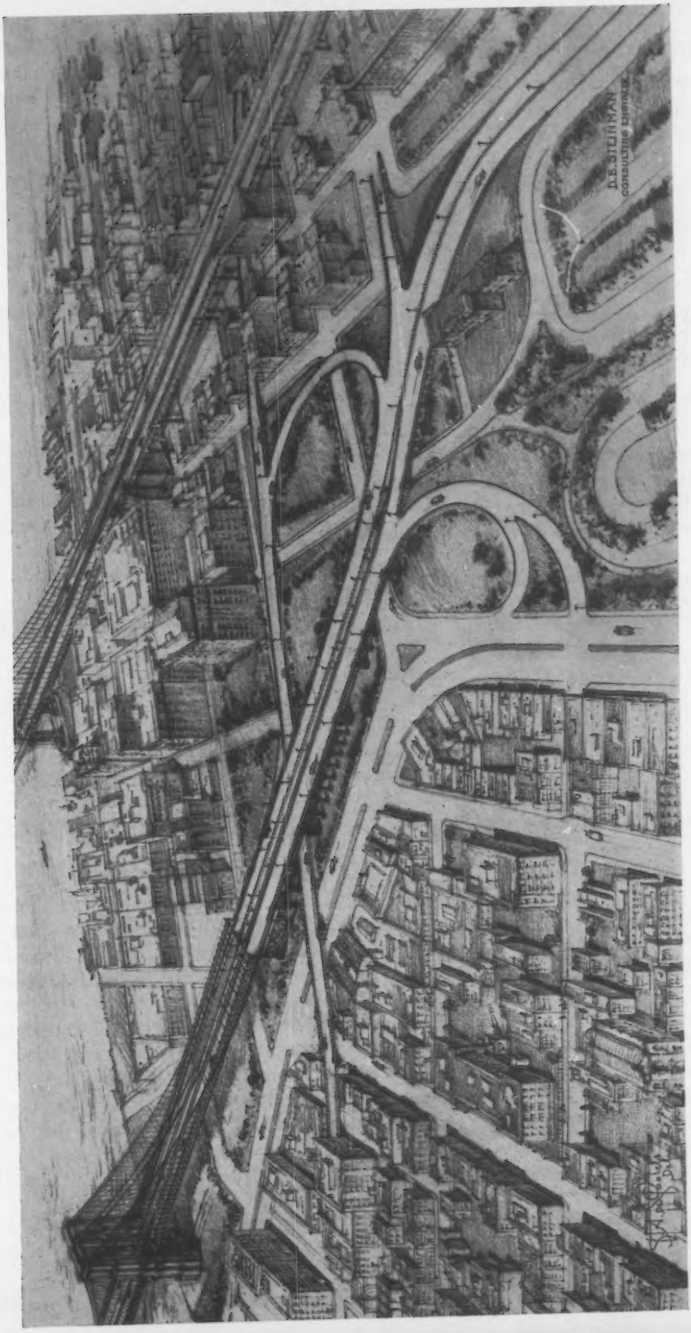


FIGURE 11. Rebuilt Brooklyn approach.

Acknowledgments

The preparation of the contract plans and specifications was done under the writer's direction for the Department of Public Works, headed by Commissioner F. H. Zurmuhlen who took a keen personal interest. In the writer's organization, the work was in charge of Mr. J. London, with cooperation of Messrs. W. E. Joyce, R. M. Boynton, and C. H. Gronquist.

CONFERENCES HELD

SECTION OF BIOLOGY

"MECHANISM OF CORTICOSTEROID ACTION IN DISEASE PROCESSES"

OCTOBER 3 AND 4, 1952

The Section of Biology held a Conference on "Mechanism of Corticosteroid Action in Disease Processes." Doctor Oscar Hechter and Doctor Gregory Pincus, The Worcester Foundation for Experimental Biology, Shrewsbury, Mass., were the Conference Co-Chairmen in charge of the meeting.

The program consisted of the following papers:

Friday, October 3

VASCULAR SYSTEM

Morning Session. Chairman: E. Shorr.

"The Influence of the Adrenal Cortex on the Behavior of the Terminal Vascular Bed," B. W. Zweifach, E. Shorr, and M. M. Black, New York Hospital, New York, N. Y.

"Effect of Cortisone on the Vascular Reactions to Certain Allergic States," W. R. Barclay, the University of Chicago, Chicago, Ill.

"The Relationship of Potassium in the Regulation of Blood Pressure with Special Attention to Corticosteroid Hypertension," S. C. Freed, R. H. Rosenman, and M. Friedman, Mount Zion Hospital, San Francisco, Calif.

"Direct Observation of the Circulation in the Hamster Cheek Pouch in Adrenal Insufficiency and Experimental Hypercorticalism," L. C. Wyman, G. P. Fulton, and M. H. Shulman, Boston University, Boston, Mass.

"Brain Blood Flow Oxygen Consumption and Electrical Activity in Relation to the Adrenal Cortex," H. Hoagland, and J. R. Bergen, The Worcester Foundation for Experimental Biology, Shrewsbury, Mass.

Reviewer: R. Levine, Michael Reese Hospital, Chicago, Ill.

CONNECTIVE TISSUES

Afternoon Session. Chairman: W. Bauer.

"Cortisone as an Antagonist of Growth Hormone and Desoxycorticosterone in Tissue Reactions," M. Taubenhaus, Michael Reese Hospital, Chicago, Ill.

"Evaluation of Structural and Chemical Changes in Connective Tissue Components," J. Gross, Massachusetts General Hospital, Boston, Mass.

"Action of Corticosteroids on Certain Components of Fibroelastic Connective Tissue," B. L. Baker, University of Michigan Medical School, Ann Arbor, Michigan.

"The Action of Steroids upon the Ground Substance as Evaluated by Permeability Studies," J. Seifter, W. E. Ehrich and D. H. Baeder, Wyeth Institute of Applied Biochemistry and University of Pennsylvania, Philadelphia, Pa.

Reviewer: A. Dorfman, University of Chicago School of Medicine, Chicago, Ill.

Saturday, October 4

INFLAMMATION AND IMMUNOLOGIC PROCESSES

Morning Session. Chairman: Valy Menkin.

"Changes in the Morphology and Permeability Produced by Corticosteroids in the Egg of Chaetopterus Pergamentaceus," L. M. Leonard and D. D. Rutstein, Havard University Medical School, Cambridge, Mass.

"Interruption by ACTH of Leukocytic Cycles in Acute Inflammation in Man," J. W. Rebuck, Henry Ford Hospital, Detroit, Mich.

"Inhibition of the Phenomenon of Local Tissue Reactivity by Corticosteroids, Salicylates and Compounds Related to Salicylates," G. Shwartzman and S. S. Schneirerson, Mount Sinai Hospital, New York, N. Y.

"Effects of Corticosteroids upon Lymph Nodes," E. H. Kass and M. I. Kendrick, Boston City Hospital, Boston, Mass.

"Suppression by Cortisone of Acquired Re-

sistance to Homologous Tissue Grafts," H. C. Stoerk, Merck Institute for Therapeutic Research, Inc., Rahway, N. J.

Reviewer: T. F. Dougherty, University of Utah School of Medicine, Salt Lake City, Utah.

BACTERIOLOGIC AND VIROLOGICAL ASPECTS

Afternoon Session. Chairman: F. Duran-Reynals.

"Effect of Cortisone upon Infectious Processes," H. J. Robinson, Merck Institute for Therapeutic Research, Inc., Rahway, N. J.

"Pneumococcal and Influenza Viral Infections," M. Finland, E. H. Kass, and M. M. Lundgren, Boston City Hospital, Boston, Mass.

"Effects of Cortisone on Tuberculosis and Pseudotuberculosis," C. A. LeMaistre, R. Tompsett, and W. McDermott, New York Hospital, New York, N. Y.

"The Effect of Cortisone and ACTH on the Pathogenesis of Tuberculosis," M. B. Lurie, P. Zappasodi, A. M. Dannenberg, Jr., and E. Gardona-Lynch, Henry Phipps Institute, University of Pennsylvania, Philadelphia, Penna.

"Poliomyelitis Infection by Parenteral Routes Made Possible by Cortisone," G. Shwartzman and S. M. Aronson, Mount Sinai Hospital, New York, N. Y.

Reviewer: L. Thomas, University of Minnesota Medical School, Minneapolis, Minn.

Round table discussion. Board of Experts: D. J Ingle, H. Selye, M. Tausk, C. N. H. Long, G. Pincus, and O. Hechter.

"GROWTH OF PROTOZOA"

OCTOBER 24 AND 25, 1952

The Section of Biology held a Conference on "Growth of Protozoa." Doctor S. H. Hutner and Doctor William Trager of New York, N. Y., were the Conference Co-Chairmen in charge of the meeting.

The program consisted of the following papers:

Friday, October 24

Morning Session. Chairman: R. P. Hall.

"Observations on *Euglena*," W. R. Robbins, Annette Hervey, and Mary E. Stebbins, New York Botanical Garden, New York, N. Y.

"Some Studies on Growth and Mating in *Chlamydomonas reinhardi*," Ruth Sager and Sam Granick, the Rockefeller Institute for Medical Research, New York, N. Y.

"Action of Light on the Formation of *Chlamydomonas* Flagella," R. A. Lewin, Maritime Regional Laboratory, Halifax, Nova Scotia.

"Vitamin Requirements of Some Marine and Freshwater Planktonic Flagellates," L. Provasoli and Irma J. Pintner, Haskins Laboratories, New York, N. Y.

"Preliminary Studies on the Response of a Chrysomonad to Vitamin B_{12} and Related Substances," F. W. Barber, D. L. Baile, C. B. Troescher, and C. N. Huhtanen, National Dairy Research Laboratories, Oakdale, Long Island, N. Y.

"Electron Microscope and Spectrophotometric Studies of a Chrysomonad and *Euglena gracilis* Grown in Light and Darkness," J. J. Wolken, E. G. Pallade, and K. R. Porter, Rockefeller Institute for Medical Research, New York, N. Y.

"Carbon Dioxide Fixation by *Euglena gracilis*," M. Calvin and V. H. Lynch, University of California, Berkeley, Calif.

Opening Discussion on Euglena and Chrysomonad Metabolism: G. G. Holz, University of California at Los Angeles, Calif.

Opening Discussion on Vitamin B_{12}: D. Hendlin, Merck & Co., Inc., Rahway, New Jersey.

Afternoon Session

"Nutrition of *Peranema*," Jack Storm and S. H. Hutner, Haskins Laboratories, New York, N. Y.

"Nutrition of Some Carnivorous Hypotrichs and Suctoria," D. M. Lilly, St. John's University, Brooklyn, N. Y.

"Protogen Metabolism in *Tetrahymena*," Gerald R. Seaman, the University of Texas, Medical Branch, Galveston, Texas.

"Growth Requirements of *Paramecium aurelia*, var. 4 stock 51, sensitives and killers in axenic medium," W. J. van Wagtendonk, R. L. Conner, C. A. Miller, and M. R. R. Rao, Indiana University, Bloomington, Indiana.

"Purine Utilization for Nucleic Acid Synthesis in Chrysomonads and Other Organisms," L. D. Hamilton, The Sloan-Kettering Institute for Cancer Research, New York, N. Y.

"Studies of the Influence of 2,6-Diaminopurine Upon the Content of Kappa in *Paramecium aurelia*, Variety 4," C. C. Stock, Winifred Jacobson, and Mary Williamson.

"Characteristics of Slime Molds and Acrasiales Affecting Growth in Pure Culture," A. L. Cohen and J. Sobels, Oglethorpe University, Georgia.

"Growth Requirements and Morphogenesis in

Dictyostelium," M. Sussman, Northwestern University, Evanston, Ill.

Opening Discussion on Ciliate Nutrition: W. H. Johnson, Wabash College, Crawfordsville, Ind.

Saturday, October 25

PARASITIC PROTOZOA

Morning Session: Chairman: William Trager.

A. Haemoflagellates.

"Nutrition of *Strigomonas* (*Herpetemonas*) *culicidarum*," Jean Cowperthwaite, M. M. Weber, L. Packer, and S. H. Hutner, Haskins Laboratories, N. Y.

"Enzyme Systems of the Culture Form of *Trypanosoma cruzi*", H. D. Baernstein, National Microbiological Institute, Bethesda, Md.

"African Sleeping Sickness Trypanosomes: Cultivation and Properties of the Culture Forms," D. Weinman, Yale University, New Haven, Conn.

Opening discussion: Th. v. Brand, National Microbiological Institute, Bethesda, Md.

B. Trichomonas.

"Studies on the Metabolism of *Trichomonas vaginalis*," A. B. Kupferberg, H. W. Singher, G. Lampson, L. Levy, and A. H. Romano, Ortho Research Foundation, Raritan, N. J.

"The Effect of Ribonuncleic Acid and Its Nitrogenous Constituents on the Growth of *Trichomonas vaginalis*," H. Sprince, R. Goldberg, G. Kucker, and R. S. Lowy, General Microbiological Laboratories, Elizabeth, N. J., and Ortho Research Foundation, Raritan, N. J.

Opening Discussion: J. G. Johnson, Albany Medical College, Albany, N. Y.

Afternoon Session.

C. Intestinal Amoebae

"Studies on the Cultivation of *Endamoeba histolytica* with *Trypanosoma cruzi*," B. P. Phillips, National Microbiological Institute, Bethesda, Md.

"Factors Affecting the Propagation of *Endamoeba histolytica*, *in vitro*, in the S-F Medium and in Tissue-bearing Substrate," J. G. Shaffer, The Chicago Medical School, Chicago, Ill.

"*In vitro* Growth and Metabolism of *Endamoeba histolytica*," A. M. Geiman and C. E. Becker, Harvard School of Public Health, Boston, Mass.

"The Cultivation of *Dientamoeba fragilis*," L Jacobs, National Microbiological Institute, Bethesda, Maryland.

Opening Discussion: C. W. Rees, National Microbiological Institute, Bethesda, Md.

D. Intracellular Parasites.

"Some Effects of Host Environment on the Course of Leishmaniasis in the Hamster," L. A. Stauber, Rutgers University, New Brunswick, N. J.

"Factors Affecting the Susceptibility of Erythrocytes to an Intracellular Parasite," R. B. McGhee, Rockefeller Institute for Medical Research, New York, N. Y.

"Further Studies on the Extracellular Cultivation of an Avian Malaria Parasite," W. Trager, Rockefeller Institute for Medical Research, New York, N. Y.

The Section of Biology provides conferences for active workers in the special fields of Biology.

Attendance is limited to those invited to participate in these conferences and to interested Members of the Academy.

NEW MEMBERS

Elected June 13, 1952

LIFE MEMBERSHIP

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SUSTAINING MEMBERSHIP

Aarons, Z. Alexander, M.D., Psychology. Private practice, New York, N. Y.

Agoston, Tibor, M.D., Psychiatry, Pharmacology. Psychiatrist, Columbus State Hosp., Columbus, Ohio.

Ambler, John J., M.D., General Science. Attending Neurosurgeon for Misericordia & St. Elizabeth Hospitals, New York, N. Y.

Faergeman, Poul M., M.D., Psychiatr Anthropology. Clinical Director, Child Guidance Clinic, Stamford, Conn.

Finn, Frederick Wall, M.D., Surgery. Co-ordinator Med. Educ., Greenwich Hospital, Greenwich, Conn.

Heald, Henry T., D.Eng., LL.D., Mathematics & Engineering. Chancellor, New York University, New York, N. Y.

Hurlbutt, Frank R., Jr., Obstetrics & Gynecology. Assist. Attending Obstetrics & Gynecology, Greenwich Hosp., Greenwich, Conn.

Janton, Otto Henry, M.D., Medicine, Cardiology. Assoc. Prof. of Medicine, Hahnemann Med. Coll., Philadelphia, Pa.

McAteer, Daniel A., M.D., Medicine. Consulting Surgeon, Coney Island Hospital, Brooklyn, N. Y.

McNeile, Hector J., M.D., Medicine. Director Medical Service, French Hospital, New York, N. Y.

Mallory, Robert, III, M.D., Obstetrics, Gynecology & Med. Photography. Obstetrician & Gynecologist, Rye, N. Y.
Miller, Theodore R., M.D., Cancer. Asst. Surgeon, Memorial Hosp., New York, N.Y.
Roberts, Donald L., C.E., Mathematics & Engineering. Vice President, Canadian Bechtel Ltd., Vancouver, B. C.
Simard, Albert Charles, M.D., Endocrinology. Private Practice, New York, N. Y.
Smiley, Douglas G., M.D., Surgery. Assistant Visiting Surgeon, Fordham Hospital, New York, N. Y.
Stewart, E. E., Dairy Research. President, National Dairy Products Corp., New York, N. Y.
Todd, B. J., Ph.C., Biology. Director, Ortho Research Foundation, Raritan, N. J.

ACTIVE MEMBERSHIP

Aagaard, George Nelson, M.D., Cardiovascular Disease. Dean & Professor of Medicine, Southwestern Med. School, Univ. of Texas, Dallas, Texas.
Abend, Lester I., M.D., Child Psychiatry. Resident in Child Psychiatry, Bellevue Psychiatric Hosp., New York, N. Y.
Aberle, David F., Ph.D., Anthropology. Associate Professor, Depts. of Soc. & Anthropology, Univ. of Michigan, Ann Arbor, Mich.
Acosta, Guillermo, M.D., Chemistry. Private Practice, New York, N. Y.
Adelson, Edward T., Psychology, Psychoanalysis. Private Practice, New York, N. Y.
Aldon, Jan J., Medicine. Private Practice, New York, N. Y.
Alperstein, Bernard B., M.D., Allergy. Chief of Allergy, Unity Hospital, Brooklyn, N. Y.
Alston, Casco, M.D., Medicine. Assistant in Medicine, College of P. & S., New York, N. Y.
Altman, Harry S., M.D., Pediatrics. Assistant Clinical Prof. of Pediatrics, College of P. & S., New York, N. Y.
Altman, Leon L., M.D., Psychiatry. Private Practice, New York, N. Y.
Arcieri, John P., M.D., Internal Medicine. Chief Cardiologist, New York Parkway Hosp., New York, N. Y.
Balagot, Reuben C., M.D., Anesthesia, Pharmacology, & Physiology. Research Fellow in Anesthesiology, University of Illinois, Research Hosp., Chicago, Ill.
Beck, William Samson, M.D., Biochemical Studies in Leukemia. Chief, Hematology Section, Atomic Energy Project, Los Angeles, Cal.
Bennett, Ivan L., M.D., Infectious Diseases. Assistant Professor of Medicine, Yale University School of Medicine, New Haven, Connecticut.
Beyler, Arthur L., Ph.D., Endocrinology. Head, Endocrinological Research, Sterling-Winthrop Research Inst., Rensselaer, N. Y.
Bivins, James Albert, D. V. M., Poultry Disease-Virus Research. Assistant Professor & Assistant Res. Specialist in Poultry Pathology, New Jersey Agricultural Exp. Station, New Brunswick, N. J.
Blewett, John P., Ph.D., Physics. Senior Physicist, Brookhaven National Lab., Upton, L. I., N. Y.
Blocker, T. G., Jr., M.D., Plastic Surgery. Prof. of Plastic and Maxillofacial Surgery, Medical Branch Hosp., University of Texas, Galveston, Texas.
Blumberg, Arnold G., M.D., Internal Medicine. Research Fellow, N. Y. U. Div., Goldwater Memorial Hosp., New York, N. Y.
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Borzelleca, Joseph Francis, Physiology, Pharmacology. St. Joseph's College, Philadelphia, Pa.
Boyd, Robert Neilson, Ph.D., Organic Chemistry. Ass't. Prof. of Chemistry, New York Univ., New York, N. Y.
Bravo, Luis de Greiff, Mathematics & Engineering. Professor of Civil Engineering, Escuela de Minas, Medellin, Colombia.
Broida, Herbert P., Ph.D., Spectroscopy. Research Physicist, National Bureau of Standards, Washington, D. C.
Bruger, Maurice, M.D., Internal Medicine and Biochemistry. Associate Prof. of Medicine, N. Y. U. Post Graduate Medical School, New York, N. Y.
Bruyn, Henry B., M.D., Pediatrics and Infectious Diseases. Assistant Professor, University of California, School of Medicine, San Francisco, Cal.
Buchanan, Robert E., Ph.D., Bacteriology. Research Prof., Agricultural Admin. Iowa State College, Ames, Iowa.
Burford, M. Gilbert, Ph.D., Physical and Analytical Chemistry. Prof. of Chemistry Wesleyan University, Middletown, Conn.
Carlson, William S., Ph.D., LL.D. (Hon.), Geology. President, State University of New York, Albany, N. Y.
Carmichael, Hattie Mae, Ph.D., Biochemistry. Associate Professor of Education, New York University, New York, N. Y.
Ciancimino, Frank E., M.D. General Surgery. Private Practice, Nyack, N. Y.
Cohen, Harry, M.D., D.Sc., Surgery. Surgeon, Columbus Hospital, New York, N. Y.
Cohn, Victor Hugo, Jr., B.S., Lehigh University, Bethlehem, Pa.
de Veer, J. Arnold, M.D., Pathology. Pathologist, The Brooklyn Hosp., Brooklyn, N. Y.
Doolan, Paul D., M.D., Endocrinology. Junior Medical Officer, MC, USN, USS Macon.
Ellis, Frank W., M.D., Cancer Research and Psychology. Chief of Oncology Dept. Veterans Administration Hospital, Long Beach, Cal.
English, James, Jr., Ph.D., Chemistry. Assistant Professor of Chemistry, Yale University, New Haven, Conn.
Erger, Benjamin D., M.D., Dermatology & Syphilology. Private Practice and Clinical Instructor, N. Y. Skin & Cancer Unit, New York, N. Y.
Evans, Edwin V., M.A., Vitamin Metabolism, Vitamin Assays. Assistant Professor, Dept. of Nutrition, Ontario Agriculture College, Guelph, Ontario, Canada.
Fanoni, Victor C., M.D., Ear, Nose, and Throat. Private Practice, Port Chester, N. Y.
Farr, Lee E., M.D., Medicine. Chairman of Medical Dept. and Physician in Chief of Hospital, Brookhaven National Lab., Upton, L. I., N. Y.
Feldmann, Robert J., M.D., Medicine. Ass't. Medical Dir., The Armour Laboratories, Chicago, Ill.
Flesch, Peter, M.D., Ph.D., Physiology and Chemistry of Skin. Assistant Prof. of Research in Dermatology, University of Pennsylvania, Philadelphia, Pa.
Flocks, Rubin H., M. D., Medicine & Biology. Professor of Urology, University of Iowa College of Medicine, Iowa City, Iowa.
Fowler, Richard G., Ph.D., Physics. Associate Professor of Physics, University of Oklahoma, Norman, Oklahoma.
Fly, Lillian Beaman, M.S., Botany & Bacteriology. Instructor in Bacteriology, University of Miami, Miami, Fla.
Frajola, Walter J., Ph.D., Biochemistry. Assistant

Professor, Dept. of Physiological Chemistry, Ohio State University, Columbus, Ohio.

French, Cyrus Edwin, Ph.D., Biochemistry and Physiology of Nutrition. Associate Professor of Nutrition, Pennsylvania State College, State College, Pa.

Greenberg, Pearl, Ph.D., Personality. Staff Psychologist, V. A. Mental Hygiene Clinic, Newark, N. J.

Guinivan, Thomas Wayne, B.S., Antibiotics. Assistant Dir. Training Dept., Antibiotic Div., Chas. Pfizer & Co., Brooklyn, N. Y.

Hart, E. Ross, Ph.D., Pharmacology. Chief, Neurology Branch, Chemical Corps Med. Laboratories, Army Chemical Center, Maryland.

Hayes, Mark A., M.D., Ph.D., Surgical Endocrinology & Metabolism. Assistant Professor of Surgery, University of Michigan Hospital, Ann Arbor, Mich.

Higgons, Reginald A., M.D., Nutrition, Convalescence, Rheumatic Heart. Dir. of Convalescent Research, St. Luke's Hosp., New York, N. Y.

Howe, Howard Atkinson, M.D., Poliomyelitis & Related Virus Diseases. Adjunct Prof. of Epidemiology, Johns Hopkins University, Baltimore, Maryland.

Jacobs, Alvin D., B. A., Biology and Medicine. N. Y. U., New York, N. Y.

Jones, Laurence, M.D., Orthopedic Surgery. Beverly Hills, Cal.

Keys, Ancel, Ph.D., Nutrition, Human Biology. Prof. and Dir. Laboratory of Physiological Hygiene, University of Minnesota, Minneapolis, Minn.

Knisely, Melvin H., Ph.D., Anatomy. Chairman and Prof. Dept. of Anatomy, Medical College of South Carolina, Charleston, S. C.

Koenig, Samuel, Ph.D., Sociology & Anthropology. Assistant Prof. of Soc. & Anthr., Brooklyn College, Brooklyn, N. Y.

Koller, Fortune, Ph.D., Mathematics and Engineering. President Gen. Armorial Universel, Bruxelles, Belgium.

Labecki, Thaddeus D., M.D., Cardiovascular Diseases. Supervisor & Medical Consultant to the Heart Disease Control Unit, Jackson, Miss.

Lasdon, Stanley S., B. S., LL.B., Chemistry. Executive Vice President, Nepera Chemical Co., Inc., Yonkers, N. Y.

Lee, C. Marshall, Jr., M.D., Pediatric Surgery. Assistant Professor of Surgery, University of Cincinnati, Cincinnati, Ohio.

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Livingood, Clarence S., M.D., Dermatology. Prof. and Chairman Dept. of Dermatology Univ. of Texas School of Medicine, Galveston, Texas.

Lutz, Brenton Reid, Ph.D., Physiology. Professor of Biology, Boston Univ., Boston, Mass.

Lynch, S. John, B.S.A., Tropical Horticulture. Dir. of Exp. Farm & Res. Prof. of Applied Tropical Botany, University of Miami, Miami, Fl.

McGuire, Carson, Ph.D., Biology, Anthropology, Psychology. Prof. of Educ. Psychology, University of Texas, Austin, Texas.

McLean, Preston G., M.D., Renal Physiology & Disease. Instructor in Physiology N. Y. U. Bellevue Med. Center, New York, N. Y.

McMillan, James R., M.D., Tropical Diseases. Medical Director, Standard Vacuum Oil Co., New York, N. Y.

Maison, George, L., M.D., Pharmacology, Physiology. Dir. of Res. & Div. Riker Labs., Los Angeles, Cal.

Matthews, LeRoy William, M.D., Biochemistry, Metabolic Diseases of Childhood. Instructor in Pediatrics & Dir. of Laboratories, Babies & Children's Hosp., Cleveland, Ohio.

Mollerstrom, Jakob V. S., M.D., Internal Medicine. Head of Metabolism Res., Wenner-Gren Inst., and Chief of Diabetes Clinic in Stockholm, Sweden.

Moon-Adams, Dabney, M.D., Dermatology & Syphilology. Assistant Prof. of Dermatology & Syphilology, College of P. & S., New York, N. Y.

Moore, Francis D., M.D., Surgery. Moseley Professor, Harvard Medical School, Boston, Mass.

Murphy, Gardner, Ph.D., Psychology, Parapsychology. Dir. of Research, Menninger Foundation, Topeka, Kansas.

Musselman, Merle M., M.D., Surgery, Dir. of Surgery, Wayne Co. General Hosp., Eloise, Mich.

Naef, Paul A., Ph.D., Chemistry, Botany, Bacteriology. Technical Manager, Sandoz Pharmaceuticals, Hanover, N. J.

Ninger, Fred C., M.S., Pharmaceuticals. Dir. of Pharmacy, Warner Inst. for Therapeutic Research, New York, N. Y.

Oesterling, M. Jane, Ph.D., Biochemistry. Assistant Prof. in Physiological Chemistry, Woman's Medical College, Philadelphia, Pa.

Peck, Mordant E., M.D., Surgery. Assistant Prof. of Surgery, University of Colorado Sch. of Medicine, Denver, Colo.

Plager, Hildegarde, M.D., Virus Research. Bacteriologist, N. Y. State Dept. of Health, Albany, N. Y.

Portis, Sidney A., M.D., Gastrointestinal Med. Senior Attending Physician, Michael Reese Hosp., Chicago, Ill.

Ransohoff, Priscilla B., M.S., General Rehabilitation. Dir. Physical Therapy Dept., Monmouth Mem. Hosp., Long Branch, N. J.

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Rutstein, Leo, Chemistry. Rutstein Laboratory, Long Beach, L. I., N. Y.

Schroeder, Henry Alfred, M.D., Cardiovascular Dis. Associate Prof. of Med. Washington University School of Medicine, St. Louis, Missouri.

Schwarz, David R., M.A., Biological Chemistry. Vice President, Schwarz Labs. Mt. Vernon, N. Y.

Sequeira, Luis, Ph.D., Mycology. Parker Fellow, Harvard University, Cambridge, Mass.

Sichere, René-Marie, M.D., Assistant at The Rothschild Hosp., Paris, France.

Silbermann, Isidor, M.D., Psychoanalysis. Private Practice, New York, N. Y.

Silver, A. Gilbert, M.D., Ear, Nose, & Throat. Private Practice, New York, N. Y.

Simmons, Norman S., Ph.D., D. M. D., Biochemistry. Chief, Enzyme Chemistry Section, UCLA, Atomic Energy Project, Los Angeles, Cal.

Singer, Samuel, M.S., Microbiology. Microbiologist, Wellcome Research Labs. Brooklyn, N. Y.

Skelton, Floyd R., Ph.D., M.D., Endocrinology. Dir. of Pharmacology, Ayerst, McKenna & Harrison, Ltd., Montreal, Canada.

Snavely, John Geoffrey, M.D., Clinical Pathology & Morbid Anatomy. Dir. of Labs., Stamford Hospital, Stamford, Conn.

Sokol, Herman, Ph.D., Antibiotics. Manager, Antibiotics Res. Div. Heyden Chemical Corp., Princeton, N. J.

Starr, Saul, M.D., Chest Diseases. Clinical Ass't Prof. Pediatrics, Univ. of the State of N. Y., Brooklyn, N. Y.

State, David, M.D., Ph.D., Surgery, Assoc. Prof. of Surgery, University Hosp., Minneapolis, Minn.

Steeves, Taylor Armstrong, Ph.D., Morphogenesis in Plants. Junior Fellow of the Soc. of Fellows, Harvard Univ., Cambridge, Mass.

Stein, Isidore, M.D., Cardiology. Assist. Cardiologist, Jewish Hosp., Brooklyn, N. Y.
Steinger, Louis, M.D., Medicine. Private Practice, Brooklyn, N. Y.
Stigman, Shepherd, B. S., Chemistry. Radiochemist, Foster D. Snell, Inc., New York, N. Y.
Suna, Harry, M.D., General Medicine. Assistant Physician, Maimonides Hosp., Brooklyn, N. Y.
Szilagyi, Nelly, M.D., Cardiology. Research Fellow, St. Francis Sanatorium for Cardiac Children, Roslyn, L. I., N. Y.
Tidewell, Herbert, Ph.D., Fat Metabolism. Prof. of Biochemistry, Southwestern Medical School, Univ. of Texas, Dallas, Tex.
Trams, Eberhard Georg, B. S., Cancer Res., Chemotherapies. Res. Ass't. George Washington Univ. Cancer Clinic, Washington, D. C.
Trefts, Hazel J., M.D., Medicine. Private Practice, Rye, N. Y.
Turbes, Calvin C., Ph.D., Neurology. Assistant Prof. of Anatomy, Indiana Univ., Bloomington, Ind.
Volweiler, Wade, M.D., Liver Diseases & Physiology. Ass't Prof. of Medicine, Univ. of Washington, Seattle, Wash.
Webster, Richard Curtis, Ph.D., Endocrinology. Ass't Prof. of Anatomy, Indiana Univ., Indianapolis, Ind.
Werner, Attie Yvonne, Ph.D., Physiology. Research Ass't., State Univ. of Iowa, Iowa City, Iowa.
Wiesner, Charles K., D.Sc., Stereochemistry. Prof. of Organic Chemistry, Univ. of New Brunswick, Fredericton, N. B., Canada.
Wortman, Bernard, M. A., Physiology. Res. Ass't., Ohio State Univ., Columbus, Ohio.
Young, Alexander W., Jr., M.D., Dermatology. Physician to Out-Patients, New York Hosp., New York, N. Y.
Zoll, Paul M., M.D., Biology. Assoc. in Medicine, Harvard Med. Sch., Boston, Mass.

STUDENT MEMBERSHIP

Heckel, Robert L., Ethnology, Gen. Anthropology. Columbia Univ., New York, N. Y.
Hirshon, Jack M., Chem. Physics, Columbia Univ. New York, N. Y.
Marinovich, Mato Luko, M.D., Biology. Bellevue Med. Center, New York, N. Y.

Elected July 15, 1952

LIFE MEMBERSHIP

Schell, Herbert H., Medicine. President, Sidney Blumenthal & Co., Inc., New York, N. Y.

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Bobst, Elmer H., Cosmetics & Ethical Drugs. President, Warner-Hudnut Inc., New York, N. Y.
Elliott, John, D. Sc., Medical Research. Director of Blood Bank & Med. Res. Foundation, Dade County, Miami, Fla.
Freund, Karl, Instrument Developing for Photography & Motion Picture Processing. President, Photo Research Corp., Burbank, Cal.
Laszlo, Erno, M.D., Biology & Chemistry. President, Erno Laszlo Inst. for Scientific Cosmetology, New York, N. Y.
Meeker, Olan, M.D., Proctology, Pharmacology. Private Practice, Riverside, Conn.

ACTIVE MEMBERSHIP

Aaron, Harold, M.D., Medicine. Medical Consultant, Consumers Union, New York, N. Y.
Ackerman, Martin, M.D., Biology. Private Practice, New York, N. Y.
Alley, John Lamont, M.D., Urology. Assistant Clincial Prof. of Urology, N. Y. U. Bellevue Med. Center, New York, N. Y.
Allen, John P., Ph.D., Physiology of Fungi. Research Assistant, N. Y. Botanical Garden, New York, N. Y.
Almansi, Renato J., M.D., Psychoanaltyic Research, Psychiatry. Consulting Neuropsychiatrist, Lutheran Hosp., New York, N. Y.
Bandelin, Fred John, Ph.C., Pharmaceutical & Medicinal Chemistry. Technical Director, Flint, Eaton & Co., Decatur, Ill.
Bergier, Jacques, Engineering. Paris, France.
Billig, Harvey E., Jr., M.D., Biology. Medical Director, The Billig Clinic, Los Angeles, Cal.
Bohan, Edward M., M.D., Diabetes. Chief of Medicine, St. Francis Hosp., Wilmington, Del.
Briggs, George M., Ph.D., Nutrition, Biology. Chief, Chick Nutrition Unit, Lab. Biochem. & Nutri., Ntl. Insts. of Health, Bethesda, Md.
Cinelli, Albert, M.D., Otology, Laryngology, Rhinology. Private Practice, New York, N. Y.
Cintron-Rivera, A. Arturo, M.D., Internal Medicine, Hematology. Res. Fellow, Hematology, Univ. of Michigan, Ann Arbor, Mich.
Coller, Frederick A., M.D., Surgery, University Hosp., Univ. Mich., Ann Arbor, Mich.
Cook, Albert William, M.D., Neurological Sciences & Physiology. Senior Neurosurgical Resident, Kings County Hosp., Brooklyn, N. Y.
Cooke, Robert Anderson, M.D., Internal Medicine. Director, Inst. of Allergy, Roosevelt Hosp., New York, N. Y.
Dew, Robert Ralph, M.D., Pediatrics, Psychology, Sociology. Private Practice, Pediatrics, Kalamazoo, Mich.
Domenjoz, Robert James, M.D., Pharmacology, Exper. Medicine. Director of Inst. of Pharmacology, Univ. of Sarreland (Sarrebruck), Basle, Switzerland.
Ecker, Arthur David, M.D., Surgical Neurology. Attending Neurosurgeon. Syracuse Mem. Hosp., Syracuse, N. Y.
Englander, Owen, B. A., Sociology. Brooklyn Coll., Brooklyn, N. Y.
Felsen, Joseph, M.D., Intestinal Infections. Director Labs. & Res., Bronx Hosp., New York, N. Y.
Gitlin, Otto, M.D., Surgery. Private Practice, Jamaica, L.I., N. Y.
Greene, William Ellery, B.A., Geology, Biology. Chairman of the Board, W. E. Greene Co., New York, N. Y.
Grossman, Arthur J., M.D., Internal Medicine. Fellow in Arthritis & Metabolic Diseases, Ntl. Inst. of Health, Public Health Service, New York, N. Y.
Haas, Valentine, M.D., Pediatrics. Director of Pediatrics, Polyclinic Med. School & Hosp., New York, N. Y.
Hartmann, Heinz, M.D., Psychoanalysis, Psychiatry. President, International Psychoanalytic Society, New York, N. Y.
Haworth, Leland J., Ph.D., Physics. Director, Brookhaven Ntl. Lab., Upton, L. I., N. Y.
Iverson, Warren P., Ph.D., Microbiology. Bacteriologist, Camp Detrick, Frederick, Md.
Jacobson, S. D., M.D., Medicine, Metabolism. Med. Director, Wayne County Gen. Hosp., Eloise, Mich.
Karnaky, Karl John, M.D., Physiology of the Vagina. Endocrinology Assoc. Prof., of Clinical Gynecology, Baylor Univ. Houston, Texas.
Kass, J. Peter, Ph.D., Chemistry, Biochemistry. Di-

rector, Res. Dept., Atlas Powder Co., Wilmington, Del.

Knox, Leila Charlton, M.D., Medicine. Consulting Pathologist, New York, N. Y.

Kunkel, Paul, M.D., Pulmonary & Vascular Physiology. Chief, Med. Service, Veterans Admin. Hosp., Newington, Conn.

Kurtin, Abner, M.D., Dermatology. Sen. Clinical Assist., Mt. Sinai Hosp., New York, N. Y.

Lamb, John H., M.D., Dermatology. Private Practice, Oklahoma City, Okla.

Lampe, Isadore, M.D., Radiotherapeutic Medicine. Prof. of Roentgenology, Univ. of Mich.

Lesser, Sandra Lloyd, M.A., Chemistry, Science Writing. Writer, Med. Press Inc., New York, N. Y.

Levy, Barnet M., D.D.S., Oral Pathology. Prof. of Dentistry, Columbia Univ., New York, N. Y.

Ludeman, Clifford G., Ph.D., Physics & Chemistry. Technical Assist. to Division Manager, Texaco Development Corp., New York, N. Y.

McNeer, Gordon, M.D., Gastroenterology. Assoc. Attending Surgeon, Memorial Hosp. New York, N. Y.

Maling, Harriet F. M., Pharmacology, Physiology. Assist. Prof. of Pharmacology, George Washington Univ. Med. School, Washington, D. C.

Mangelsdorff, Arthur F., M.D., Occupational Medicine. Assist. Med. Director, American Cyanamid Co., Bound Brook, N. J.

Maral, René, M.D., Virology. Assist. faculté de médecine, inst. bactériologique, Lyon, France.

Martins, Thales, M.D., Endocrinology. Head, Dept. of Endocrinology, Oswaldo Cruz Inst., Rio de Janeiro, Brazil.

Meister, Lester, M.D., Rheumatology, Endocrinology. Section Chief in Medicine, V. A. Hosp., Long Beach, Cal.

McGuinness, Madge C. L., M.D., Meteorology. Director of Physical Medicine, Misericordia Hosp., New York, N. Y.

Melicow, Meyer M., M.D., Uropathology. Assist. Clinical Prof. Urology, College of Physicians & Surgeons, Columbia Univ., New York, N. Y.

Meyer, Milo G., M.D., Internal Medicine. Head, Dept. of Internal Medicine, Gardner Meyer Clinic, Michigan City, Ind.

Mondragon, Harold, B.S., M.D., Biology. Resident Pathologist, Passaic General Hosp., Passaic, N. J.

Moore, Arthur Ulric, Ph.D., Psychobiology. Senior Res. Associate, Cornell Behavior Farm, Dept. of Psychology, Cornell Univ., Ithaca, N. Y.

Munch, James C., Ph.D., Pharmacology, Toxicology, Internal Medicine. Med. Director, Strong Cobb & Co., Inc., Shaker Heights, Ohio.

Murdock, Harold R., Jr., Ph.D., Pharmacology. Senior Pharmacologist, Johnson & Johnson Research Foundation, New Brunswick, N. J.

Norris, Jack C., M.D., Pathology, Clinical Pathology, Hematology. Assist. Prof. Pathology, Emory Univ. (inac). President, Fulton County Med. Society. Private Practice, Atlanta, Ga.

Nyvall, Harry O. J., M.D., Medicine. Director, Hospital & Clinic (Smith), Gustine, Texas.

Ostow, Mortimer, M.D., Psychiatry & Neurology. Private Practice, New York, N.Y.

Page, Edouard, Ph.D., Fat Metabolism. Director & Prof., Inst. of Physiology, Laval University, Quebec, Canada.

Papez, James W., M.D., Medical Biology, Neurology. Director of Biological Res., Columbus State Hosp., Columbus, Ohio.

Pareira, Morton D., M.D., Surgery. Instructor in Surgery, Washington Univ., St. Louis, Mo.

Reddick, Grady, M.D., Internal Medicine. Clinical Prof. of Medicine, Univ. of Texas, Dallas, Texas.

Reynolds, Whitman Mead, M.D., Life Ins., Medicine, Biology. Associate Med. Director, Equitable Life Assur. Society, New York, N. Y.

Rosen, George M., M.D., Biology, Physics & Chemistry. Private Practice, Surgery, Kew Garden Hills, L. I., N. Y.

Rosenthal, Julius, M.D., Pathology. Pathologist, Goldwater Mem. Hosp., New York, N. Y.

Sandweiss, David J., M.D., Gastroenterology. Assoc. Physician, Internal Med., Harper Hosp., Detroit, Mich.

Santesson, Gunnar O. C. H., M.D., Internal Medicine. Physician, Medical Clinic, Royal Serafimer Hosp., Stockholm, Sweden.

Schattman, Esther Preger, M.A., B.S., Psychology. Psychologist, Ethical Culture Schools, New York, N. Y.

Schmidt-Nielsen, Knut, Ph.D., Physiology. Prof. Dept. of Zoology, Duke Univ., Durham, N. C.

Schwartz, Philipp, M.D., Pathology, Tuberculosis. Dir. Inst. of Gen. Pathology, Univ. of Istanbul, Turkey.

Scott, William E., Ph.D., Organic Chemistry. Res. Chemist, Hoffman La Roche Inc., Nutley N. J.

Sherwood, Noble P., M.D., Clinical Pathology & Immunology. Prof. of Bacteriology, Univ. of Kansas, Lawrence, Kan.

Siegel, Sigmund A., M.D., Surgery. Assist. Prof. Surgery, N. Y. Med. Coll., New York, N. Y.

Silbermann, Maximilian, M.D., Neurology, Psychiatry. Assoc. Att. Neurologist, Presbyterian Hosp., Neurological Inst., & Vanderbilt Clinic, Columbia Univ., New York, N. Y.

Simon, John L., M.D., Neurology, Psychiatry. Private Practice, New York, N. Y.

Smedresman, Sidney, M.D., Medicine, Biology, Chemistry, Psychology. Physician, N. Y. City Health Dept., New York, N. Y.

Spaet, Theodore H., M.D., Hematology. Instructor in Medicine, Stanford Univ., Med. School, San Francisco, Cal.

Sponer, Hertha D. E., Ph.D., Structure & Spectroscopy of Polyatomic Molecules. Prof. of Physics, Duke Univ., Durham, N. C.

Stern, Arthur M., Ph.D., Physiology of Fungi. Assist. Prof. of Bacteriology, Rutgers Univ., New Brunswick, N. J.

Sternberg, Harry Molmuth, M.D., Medicine, Pediatrics. Attending Ped., Greenpoint Hosp., Brooklyn, N. Y.

Stone, Irwin Kingsbury, M.D., Internal Medicine. Instructor, Internal Medicine, Cardiology, State Univ., of N. Y., Syracuse, N. Y.

Trigg, Edward G., D.V.M., Microbiology. Prof. & Chairman, Dept. of Bacteriology, Tuskegee Inst., Ala.

Watson, George F., M.D., Cancer. Private Practice, Kitchener, Ont., Canada.

Weiss, Frederick, M.D., Psychoanalysis, Member of Faculty, Amer. Inst. for Psychoanalysis, & New School, New York, N. Y.

Wells, Herschel James, M.D., Internal Medicine. Resident, Internal Medicine, Wayne County Gen. Hosp., Eloise, Mich.

Wexler, Bernard C., Ph.D., Anatomy & Physiology, Endocrinology. Investigator, Baxter Labs., Inc., Morton Grove, Ill.

Williams, Raymond C., D.V.M., Anatomy. Head, Dept. of Veterinary Anatomy, Tuskegee Inst., Ala.

Wolfman, Earl Frank, M.D., Surgery. Resident Surgeon, Univ. Hosp., Ann Arbor, Mich.

Elected August 15, 1952

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Wood, Walter Abbotte, Geography. Director, Arctic Inst. of North America, New York, N.Y.

SUSTAINING MEMBERSHIP

Ely, DeForest, M.D., Medicine. Pharmaceutical Advertising, New York, N. Y.
Scholl, William M., M.D., Orthopedics. Chicago, Ill.

ACTIVE MEMBERSHIP

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Alfonso, Rafael, M.D., Surgery. Ass. Physician, Worcester State Hosp., Worcester, Mass.
Anson, Cordelia Waller, B.A., Anthropology. New York, N. Y.
Arbeit, Sidney R., M.D., Ballistocardiography. Jersey City, N. J.
Barclay, Ralph K., Ph.D., Cancer. Assoc. Division of Exp. Chemotherapy, Sloan Kettering Inst. for Cancer Res., New York, N. Y.
Bernstein, Alfred J., B.S., Radiology. Attending Radiologist, Bronx Hospital, New York, N. Y.
Bloch, Alan, M.A., Physics. Chief Development Engineer & Vice Pres., Audio Instrument Co., Inc., New York, N. Y.
Brill, Edmund R., Ph.D., Biology. Independent Research & Writing in Biology, New York, N. Y.
Cantor, Alfred J., M.D., Proctology. Clinical Practice of Proctology, Flushing, L. I., N. Y.
Carlozzi, Michael, M.D., Medical Research. Medical Division, Charles Pfizer & Co., Brooklyn, N. Y.
Costa, Joseph A., M.D., Pediatrics. Chief of Pediatric Service, Northern Westchester Hospital, Mt. Kisco, N. Y.
Cunningham, Charles H., D.V.M., Virology. Assoc. Prof. of Bacteriology, Michigan State Coll., East Lansing, Mich.
Dean, William Hope, M.D., Internal Medicine. Malone & Hogan Clinic Hosp. Foundation, Big Spring, Texas.
De Feo, Vincent Joseph, M.S., Physiology. Res. Fellow in Physiology, Columbus, Ohio.
De Palma, Rudolph, B.S., Biology. Chemist, Armour & Co., Chicago, Ill.
D'Oronzio, Joseph B., M.D., Biology. Urologist, Columbus Hosp., New York, N. Y.
Edelman, Morton H., M.D., Tropical Medicine. Internist, Mt. Sinai Hosp., Private Practice, New York, N. Y.
Edelstein, Sidney M., B.S., History of Chemistry & Physics, Dexter Chem. Corp., New York, N. Y.
Ehrenpreis, Bernard, M. D., Radiology. Clinical Prof. of Radiology, State University of New York, Brooklyn, N. Y.
Forster, Sigmund, M.D., Physical Medicine. Associate in Physical Med., Coney Island Hosp., Brooklyn, N. Y.
Friedman, B.A., Ph.D., Plant Pathologist, New York, N. Y.
Halliburton, Arthur, Research in Abnormal Psychology. Feature Writer, King Features, New York, N. Y.
Hemke, Emily D., B.A., Endocrinology. Res. Assist., Sterling Winthrop Res. Inst., Troy, N. Y.
Hoffman, William J., M.D., Cancer. Assoc. Surgeon, Chief of Tumor Surgery, Queens Gen. Hosp., New York, N. Y.
Holman, James, M.D., Allergy. Private Practice, Dallas, Texas.
Jablon, James M., Ph.D., Bacteriology. Bacteriologist, St. Francis Sanatorium for Cardiac Children, Roslyn, L. I., N. Y.
Krainain, Joseph M., M.D., Medicine. Private Practice, Bedford, N. Y.
Kronish, Donald Paul, B.A., Microbiology. New York, N. Y.
Landman, Gerda S., M.D., Venerealogy & Dermatology. Med. Officer in charge of V. D., Gallinger Mun. Hosp., Washington, D. C.
Leveridge, Leo L., M.D., Medical Motion Pictures as Audio-Visual Aids. Medical Service Dept., Chas. Pfizer & Co., Brooklyn, N. Y.
Levin, William C., M.D., Internal Medicine. Assoc. Prof. of Internal Medicine, Univ. of Texas, Galveston, Texas.
McGowan, Lillian, M.D., Psychiatry. Private Practice, New York, N. Y.
Mandel, Manley, Ph.D., Microbiology. Guest Investigator, Haskins Labs., New York, N. Y.
Miller, Joseph S. A., M.D., Psychology, Psychiatry. Med. Director, Hillside Hosp., Glen Oaks, L. I., N. Y.
Moreton, Robert Dulaney, M.D., Radiology. Partner Tom B. Bond Radiological Group, Fort Worth, Texas.
Paullada, Juan Jose, M.D., Endocrinology. Res. Fellow, Albany Hosp., Albany, N. Y.
Perri, Giulio C., Biological Chemistry. University of Rome, Italy.
Peters, William Henry, Jr., M.S., Medical Research. Staff Writer, Lederle Labs., New York, N. Y.
Porcello, Joseph A., M.D., Physiology, Chemistry. Private Practice, Pleasantville, N. Y.
Reed, Raymond E., Chemistry. Vice President, Toni Co., Chicago, Ill.
Rinfret, Arthur Piers, Ph.D., Biochemistry. Director of Res., Victory Packing Co., Los Angeles, Cal.
Roberts, E. S., M.E., Biology, Physics, Chemistry, Maths. & Eng. Vice Pres. & Chief Engineer, Chem. Construction Corp., New York, N. Y.
Robitzek, Edward H., M.D., Phthisiology. Acting Director Medicine, Sea View Hosp., Staten Island, N. Y.
Schlyen, Samuel M., M.D., Etiology, Pathology of Cancer. Ntl. Cancer Inst., Bethesda, Md.
Schur, Max, M.D., Psychoanalysis. Clinical Assoc. Prof., Dept. of Psychiatry, State Univ. Coll. of Medicine, New York, N.Y.
Schur, Milton Oscar, B.S., Mathematics, Engineering, Ecusta Paper Corp., Pisga Forest, N. C.
Sensening, E. Carl, Ph.D., Human Embryology, Anthropology. Assoc. Prof. of Anatomy, Med. Coll. of Alabama, Birmingham, Ala.
Simmonds, Francis, B. S., Radiochemistry & Physics. Res. Technician in Physics, Mt. Sinai Hosp., New York, N.Y.
Sivel, Wenceslas Joseph, Ph.D., Engineering, Mathematics Res. Engineer,-Design. Parsons, Brinkerhoff, Hall, MacDonald, New York, N. Y.
Stamler, Jeremiah, M.D., Cardiovascular Research. Med. Res. Inst., Michael Reese Hosp., Chicago, Ill.
Taylor, Seymour, Pharmacy, Antibiotics, Biology, Psychology. Director of Training, Chas. Pfizer & Co., Brooklyn, N. Y.
Tobis, Jerome S., M.D., Medicine. Assoc. Prof., N. Y. Med. Coll., Flower & Fifth Avenue Hosp., New York, N. Y.
Turi, A. Edward, M.D., Metabolism, Antibiotics. Director, Prospect Hill Med. Clinic, Newark, N. J.

Waggener, Karl J., M.D., Psychiatry, Meteorology. Medical Director, Woodcroft Hosp., Pueblo, Colo.
Wedemeyer, Marlin R., M.D., Psychiatry. Superintendent, Columbus State Hosp., Columbus, Ohio.
Wilcox, Paul W., M.S., Pharmaceutical Res., Assist. Director of Res., Sharp & Dohme Inc., West Point, Pa.
Wilson, William A., M.D., Internal Medicine. Assoc. Physician, Hartford Hosp., Hartford, Conn.
Wood, Earl Leroy, M.D., Medicine, Surgery. Surgeon, Otolaryngology, Private Practice, Newark, N. J.

STUDENT MEMBERSHIP

O'Reilly, Hubert Ronald, Civil Engineering. Manhattan Coll., New York, N. Y.

Elected September 15, 1952

SUSTAINING MEMBERSHIP

Schwartz, George, M.D., Biology. Lecturer, Polyclinic Post-Graduate Medical School, New York, N. Y.

ACTIVE MEMBERSHIP

Abelson, Neva M., M.D., Pediatrics. Assist. Prof. of Pediatrics, Univ. of Penna., Philadelphia, Pa.
Adler, Kurt, M.D., Psychology. Private Practice, Jamaica Hospital, L. I., N. Y.
Balkin, Seymour S., M.D., Medicine, Lung Diseases. Private Practice, Forest Hills, L. I., N. Y.
Bases, Robert E., B.A., Medicine, Chemistry. N. Y. Univ., Coll. of Medicine, New York, N. Y.
Beckhard, Erwin, M.D., Biology, Anthropology. Private Practice, Forest Hills, L. I., N. Y.
Belinkin, William, M.D., Dermatology. Board of Health, Dept. Social Hygiene, New York, N. Y.
Berg, John Williams, Endocrine Pathology. Post-Graduate Fellow, Yale School of Medicine, New Haven, Conn.
Beyer, Robert Edward, M. S., Endocrinology, Biology. Grad. Assistant, Brown Univ., Providence, R. I.
Blackford, Virginia L., M.S., Bacteriology. Bacteriologist, Walter Reed Army Medical Center, Washington, D. C.
Blankmeyer, Harrison C., B.S., Scientific Director, Owens Corning Fiberglas Corp., Newark, Ohio.
Brandman, Leonard J., M.D., Obstetrics & Gynecology, Endocrinology. Private Practice, Flushing, L. I., N. Y.
Bucalo, Harry D., Jr., M.D., Pathology. U. S. Air Force, Maxwell, Ala.
Carson, Arthur Henry, B.A., F.I.C.S., M.D., Urology. Private Practice, Elmhurst, L. I., N. Y.
Coltof, Louis A., M.D., Medicine. Private Practice, Ozone Park, L. I., N. Y.
Dittmer, Herbert Gerard, M.D., Medicine. Assist. Surgeon, Lutheran Hospital, New York, N. Y.
Ehrlich, Harry, M.D., Biology, Physics, Chemistry. Assist. Attending Obstetrics, Caledonia Hosp., Brooklyn, N. Y.
Epstein, Bernard S., M.D., Radiology. Assoc. Radiologist, Jewish Hosp., of Brooklyn, N. Y.
Fleck, Henry, M.D., Medicine. Private Practice, New York, N. Y.
Fradkin, William Z., M.D., Diarrheal Diseases. Assoc. Bacteriologist, Dept. of Labs., Jewish Hosp. of Brooklyn, N. Y.
Friedland, Lester M., M.D., Pathology. Director of Labs., Holyoke Hosp., Holyoke, Mass.
Geppert, Leo J., M.D., Chemistry, Medicine. Chief, Pediatric Service, Brooke Army Hospital, Fort Sam Houston, Texas.
Gillet, Alfred, Chemistry. Professor, Applied Organic Chemistry-Electrochemistry, University of Liège, Belgium.
Glaz, A. Andre, M.D., Psychology. Private Practice, New York, N. Y.
Gulotta, Gasper A., M.D., Cardiological Research. Assist. Administrator, St. Francis Sanatorium, Roslyn, L. I., N. Y.
Holysz, Roman P., Ph.D., Organic Chemistry. Research Chemist, Upjohn Co., Kalamazoo, Mich.
Jacobs, Leon, Ph.D., Parasitic Protozoa. Scientist, U. S. Public Health Service, Ntl. Insts. of Health, Bethesda, Md.
Kara, Gerald B., M.D., Ocular Histopathology. Practicing Ophthalmologist, New York, N. Y.
Kovitz, Benjamin, M.D., Psychiatry. Clinical Director, Columbus State Hospital, Columbus, Ohio.
Kramer, Norman C., M.S., Biochemistry, Medicine. Medical Student, Consultant in Biochemistry & Research, Veterans Admin. Center, Martinsburg, West Va.
Kuizumi, Kiyomi, M.D., Physiology. Instructor, State Univ. of N. Y., Brooklyn, N. Y.
Lee, Yin Chen, M.D., Pathology. Instructor, State Univ. of N. Y., Brooklyn, N. Y.
Lehrer, Harold, M.D., Medicine, Physics. Intern, Williamsburgh General Hospital, Brooklyn, N. Y.
Liberman, Jerrold S., M.D., Internal Medicine. Instructor, Cornell Univ. Med. Coll., New York.
Martin, William B., D.D.S., Oral Surgery. Assist. Prof. of Oral Surgery, Loyola Univ., Dental School, Chicago, Ill.
Miller, Burdsall D., M.S., Bacteriology. Assist. Chief, Tactics & Techniques Sect., U. S. Air Force, Washington, D. C.
Miller, George, B.S., Physical Chemistry. Senior Chemist, Stauffer Chemical Co., Chauncey, N. Y.
Newburger, Maurice, Ph.D., Clinical Psychology. Director, Juvenile Res., Dept. of Public Welfare, State of Ohio, Columbus, Ohio.
Pai, K. V. Srinivasa, M.S., Biochemistry, Botany. Res. Fellow, Gov. of India, Pallavaram, India.
Pixley, Edna E., B.A., Cancer Research. Chief, cytologist, State Univ. of Iowa, Iowa City, Iowa.
Revere, Althea, M.S., Microscopy. Assoc. Prof. & Director, Electron Microscope Lab., Stevens Inst. of Technology, Hoboken, N. J.
Rodnan, Gerald P., M.D., Hepatic Disorders. Resident Physician, Duke Univ. Hosp., Durham, N. C.
Rosenthal, Eugene J., M.D., Biochemistry. Physician, Montefiore Hosp., New York, N.Y.
Shapiro, Lawrence M., M.D., Biology, Psychology, Anthropology. Practicing Pediatrician, New York, N. Y.
Shapiro, Philip, M.D., Medicine, Biology, Anthropology, Psychology. Private Practice, New York, N. Y.
Stanley, Edward A., Geology & Biology. Lab. Tech., Medical, Middlesex Gen. Hosp., New Brunswick, N. J.
Schneer, Henry I., M.D., Psychological Medicine. Professor in Psychiatry, Adelphi Coll., Garden City, L. I., N. Y.
Steinetz, Bernard G., Jr., B.A., Endocrinology. Teaching Assist., Rutgers Univ., New Brunswick, N. J.
Stout, Helen Anne, Ph.D., Microbiology. Res. Assoc., E. R. Squibb & Sons, New Brunswick, N. J.
Van Cott, Harold Porter, Ph.D., Psychology. Assist. Prof. of Psychology, Alfred Univ., Alfred, N. Y.
Walker, D. B., M.D., Surgery. Private Practice, Rochester, N. Y.

Watman, Robert N., M.D., Physiology, Exp. Surgery. Resident in Surgery, Ohio State Univ. Hosp., Columbus, Ohio.
Wiesel, Leon Lawrence, M.D., Arthritis. Clin. Assist. Prof. of Medicine, N. Y. S. Univ., Coll. Med., Brooklyn, N. Y.
Wilson, John, M.D., Biology, Internal Medicine. Director of City-County Dept. of Health, Ashland, Kentucky.
Young, I. Henry, M.D., Gastroenterology. Private Practice, Irvington, N. J.
Yu, Paul N. G., M.D., Internal Medicine. Instructor & Res. Fellow, Univ. of Rochester, N. Y.

Elected October 23, 1952

SUSTAINING MEMBERSHIP

Bizzare, Emanuel V., M.D., Medicine, Surgery. Private Practice, Long Island City, N. Y.
Borken, Norman, M.D., Physical Medicine. Resident, Physical Med. & Rehabilitation, Mt. Sinai Hosp., New York, N. Y.
Nichols, Joseph, Ph.D., Organic Chemistry. Chief, Div. of Organic Chemistry, Ethicon Suture Labs., Inc., Princeton, N. J.

ACTIVE MEMBERSHIP

Alpert, Morton, Ph.D., Endocrinology. Instructor in Anatomy, Ohio State Univ., Columbus, Ohio.
Apogi, Evelyn, M.D., Anesthesiology. Director of Anesthesiology, Anesthes. Assoc. of Flushing, L. I., N. Y.
Barnard, Robert D., M.D., Antibiotics. Private Practice, Laurelton, L. I., N. Y.
Barraclough, Charles A., Jr., Endocrinology. Rutgers Univ., New Brunswick, N. J.
Bate, Doris, M.D., Radiology. Fellow, American Cancer Society, New York, N. Y.
Becker, Ernest Lovell, M.D., Renal Physiology. Instructor, Dept. of Physiology, N. Y. U. Bellevue Medical Center, New York, N. Y.
Behm, Juan J., M.A., Geology, Mineralogy. Assist. Science Curator, Staten Island Inst. of Arts & Sciences, N. Y.
Beneventi, Francis A., M.D., Urology. Surgeon, New York, N. Y.
Benzer, Paul, D.D.S., Experimental Marine Biology. Instructor, Oral Surgery, N. Y. U., New York, N. Y.
Blankfein, J., M.D., Surgery. Assist. Physician, Queens General Hospital, N. Y. C.
Blaustein, Ancel Usher, M.D., Hematology. Director Labs., Bayonne Hosp., N. J.
Block, Marvin A., M.D., Anesthesia. Private Practice, Buffalo, N. Y.
Breslow, Samuel, M.D., Medicine. Private Practice, Perth Amboy, N. J.
Brill, A. Bertrand, B.A., Physical Biochemistry. Dept. of Pediatrics, Univ. of Utah, Salt Lake City, Utah.
Chan, Kam-Fai, M.D., Parasitology. Res. Assoc. in Parasitology, Columbia Univ., New York, N. Y.
Cohen, Herbert G., M.D., Orthopedic Surgery. Instructor, N. Y. Medical Coll., Flower & Fifth Avenue Hosp., New York, N. Y.
Cohen, Theodore, M.D., Internal Medicine. Resident in Internal Medicine, Bellevue Hospital, New York, N. Y.
Cook, George Crouse, B.S., Mathematics. N. Y. Stock Exchange, New York, N. Y.
Coulston, Frederick, Ph.D., Pathology. Director of Pathology, Sterling Winthrop Res. Inst., Rensselaer, N. Y.
Davis, Robert K., Ph.D., Radiation Biology. Section Chief Zoology Section, Biological Res. Div., Mount Lab., Monsanto Chem. Co., Miamisburg, Ohio.
Deschiens, Maurice V. P., Chemistry. Chemical Engineer, New York, N. Y.
Drabkin, Bernard, B.A., Medicine. Med. Student, Univ. of Chicago, Ill.
Dworetzky, Murray, M.D., Allergy, Immunology. Instructor in Medicine, Cornell Univ., Med. Coll., New York, N. Y.
Eckert, William G., M.D., Pathology. Intern in U. S. Pub. Health Service, Marine Hosp., Staten Island, N. Y.
English, James Andrew, D.D.S., Oral Pathology. Head, Dental Division, Naval Med. Res. Inst., Bethesda, Md.
Fanslau, Charles E., D.V.M., Veterinary Medicine. Director of Vet. Div., Winthrop Stearns Inc., New York, N. Y.
Fazio, Michael G., M.D., Medicine. Private Practice, Brooklyn, N. Y.
Feinberg, Harry, M.D., Surgery. Private Practice, Brooklyn, N. Y.
Feitell, Arthur, M.D., Proctology. Chief, Proc. Stuyvesant Clinic, New York, N. Y.
Ferreol, Ernesto V., M.D., Pathology. Resident in Pathology, Jewish Hosp., of Brooklyn, N. Y.
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Fink, Frederick C., Ph.D., Bacteriology. Coordinator, Hosp. Lab. Advisory Service, Chas. Pfizer & Co., Brooklyn, N. Y.
Frankel, David Allan, M.D., Obstetrics & Gynecology. Obstetrician & Gynecologist, Brooklyn, N. Y.
Gais, Elmer S., M.D., Metabolic Diseases. Assoc. Prof. of Clin. Med., N. Y. U. Post Grad. School of Med., New York, N. Y.
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Gilston, Robert J., M.D., Clinical Oncology. Albany Med. Coll., Albany. N. Y.,
Good, Robert A., Ph.D., Pediatrics. Assist. Prof. of Pediatrics, Univ. of Minnesota, Minneapolis, Minn.
Goldberg, Bernard, M.D., Ophthalmology. Res. Physician, N. Y. Eye & Ear Hosp., New York, N. Y.
Goldman, Stanford, Ph.D., Maths. Physics. Prof. of Electrical Engin., Syracuse Univ., H. Y.
Goodof, Irving I., M.D., Pathology. Pathologist, Thayer Hosp., Waterville, Maine.
Gorelick, Arthur N., D.V.M., Med. Bacteriology, Camp Detrick, Frederick, Md.
Haskins, Edna Ferrell, Ph.D., Genetics. Senior Staff Member, Haskins Labs., New York, N. Y.
Hodes, Horace L., M.D., Research, Biology. Pediatrician, Mt. Sinai Hosp., New York, N. Y.
Holtkamp, Dorsey E., Ph.D., Endocrinology. Senior Res. Scientist, Smith, Kline & French Labs., Philadelphia, Pa.
Keating, John H., M.D., Medicine. Director of Medicine, St. Luke's Hosp., New York, N. Y.
Kellen, Ernest Alfred, M.D., Neuropsychiatry. Staff Physician, Columbus State Hosp., Columbus, Ohio.
Kish, Ernest F., M.D., Biology. Physician & Surgeon, N. W. Hosp., Mt. Kisco. N. Y.
Klein, Mordon, Ph.D., Microbiology. Assoc. Prof., of Microbiology, Temple Univ., Philadelphia, Pa.
Koch, Henry J., Jr., M.D., Clinical Investigation. Director, Dept. of Clinical & Exp. Hematology, Memorial Center, New York, N. Y.
Kornfield, Alfred T., B.A., Physiology. Res. Fellow, Dept. of Physiology, Ohio State Univ., Columbus, Ohio.

Kraght, Peter Ervin, M.S., Meteorology. Assist. Supv. Flight Dispatch, American Air Lines, La Guardia Field, N. Y.

Krivis, Alan Frederick, B.A., Chemistry. Chemist, Sloan Kettering Inst., New York, N. Y.

Landgraf, John L., Ph.D., Anthropology. Assist. Prof., N. Y. U., New York, N. Y.

Litsky, Warren, Ph.D., Bacteriology. Assist. Res. Prof. Dept. of Bacteriology, Univ. of Mass., Amherst, Mass.

Lynch, John E., Ph.D., Bacteriology. Res. Bacteriologist, Pfizer Therapeutic Inst., Maywood, N. J.

Manthey, John A., M.S., Biology. Analytical Chemist, Cleveland, Ohio.

Mason, Richard C., Ph.D., Endocrinology. Res. Assoc. Merck Inst., Rahway, N. J.

Millman, Samuel, M.D., Internal Medicine. Assoc. Attending Physician, Jewish Hosp., Brooklyn, N. Y.

Nagaki, Daizo, M.D., Bacteriology. Visiting Doctor, Dept. of Bacteriology, Cornell Univ., New York, N. Y.

Pincus, Irwin J., M.D., Metabolism. Assist. Prof. Physiology, Jefferson Med. Coll., Philadelphia, Pa.

Roberts, John L., Ph.D., Physiology. Instructor, Univ. of Mass., Amherst, Mass.

Rosenberg, Bernard, B.S., Biology. Director, Stamford Med. Lab., Stamford, Conn.

Salgado, Ernesto D., M.D., Endocrinology. Res. Assist. Institut de Médicine et de Chirurgie expérimentales, University of Montreal, Canada.

Santelli, Salvatore, B.S., Pharmacy. Assist. Prof. Pharmacy, Brooklyn Coll. of Pharmacy, Brooklyn, N. Y.

Schall, Herbert M., Ph.D., Clinical Psychology. Private Practice, New York, N. Y.

Schneider, Elias, M.D., Psychiatry. Res. Assist. in Psychiatry, Columbia Univ., New York, N. Y.

Schreiber, George, M.D., Biology. Clinical Assist. Surgery, Beekman-Downtown Hosp., New York, N. Y.

Schulert, Arthur Robert, Ph.D., Biochemistry. Res. Assoc. N. Y. U., Res. Service, Goldwater Mem. Hosp., New York, N. Y.

Schullinger, Rudolph N., M.D., Biology. Assist. Prof., Columbia Univ., Physicians & Surgeons, New York, N. Y.

Schulman, Irving, M.D., Pediatrics, Hematology. Instructor in Pediatrics, Cornell Univ., Med. Coll., N. Y. C.

Schultz, Lester J., M.D., Surgery. Private Practice, New York, N. Y.

Schwarz, Alfred W., M.D., Hematology, Pathology. Assoc. Hematologist, Jewish Mem. Hosp., New York, N. Y.

Segal, Benjamin, M.D., Obstetrics. Private Practice, & Lincoln Hosp., New York, N. Y.

Seidlin, Samuel, M.D., Endocrinology, New York, N. Y.

Seley, Abbey David, M.D., Gynecology & Obstetrics. Beth Israel Hosp., New York, N. Y.

Seymour, Francis I., M.D., Gynecology. Private Practice, New York, N. Y.

Sher, Joseph Jerome, M.D., Radio Isotopes. Radiation Therapist, Brooklyn, N. Y.

Shettles, Landrum Brewer, B.A., Obstetrics. Assoc. in Obstetrics, Columbia-Presbyterian Med. Center, New York, N. Y.

Shiner, Irving, M.D., Medicine. Private Practice, New York, N. Y.

Silverstein, Harold, M.A., Physiology. Teacher, Benjamin Franklin High School, New York, N. Y.

Sinkoff, Marvin Wolfe, M.D., Endocrinology. Fellow, Dept. of Pharmacology, N. Y. Univ. Coll. of Med., New York, N. Y.

Snyder, H. Blake, Protein Nutrition. Human Engineering Foundation, New York, N. Y.

Stanfield, C. Alpheus, B.A., Biology. Univ. of Rochester, N. Y.

Surchin, Harold P., M.D., Medicine, Psychiatry. Senior Res. in Psychiatry, Kings County Hosp., Brooklyn, N. Y.

Thaysen, Jorn C. Hess, M.D., Physiology of Secretion. Rockefeller Inst. for Med. Res., New York, N. Y.

Vassar, Philip Stanley, L.R.C.P., M.R.C.S., Blood Resident in Pathology, Columbia-Presbyterian Med. Center, New York, N. Y.

Wallach, Jacques B., M.D., Pathology. Assist. Pathologist, Queens Gen. Hosp., N. Y.

STUDENT MEMBERSHIP

Michard, Edward C., Jr., B.S., M.A., Physiology. New York Univ., Coll. of Dentistry, New York, N. Y.